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DERMATOMYOSITIS

A STUDY OF FORTY CASES

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Dermatomyositis, or primary polymyositis, is a rare disease of multiform character. The chief anatomic features of this syndrome are nonsuppurative inflammation and degeneration of many muscles or even of the entire skeletal musculature. The condition may set in acutely, with severe and rapidly progressive symptoms, or it may appear in comparatively chronic form, with mild symptoms and sometimes with complete or partial remissions. In any event, the outcome frequently is fatal, whereas recovery, when it occurs, is likely to be marked by residual muscular atrophy and permanent cutaneous changes.

Between 1926 and 1939 40 cases of subacute and chronic dermatomyositis were observed at the Mayo Clinic. They constitute a group from which all doubtful, abortive or localized instances have been excluded. This study comprises an analysis of the unquestionable cases, including a review of the history and subsequent course, so far as we have been able to trace them.

CLINICAL AND LABORATORY FEATURES

A history of acute infection antedating the onset, chiefly tonsillitis, influenza and sinusitis, was elicited in 11 cases. Recent parturition preceded the disease in 3 cases. Chronic diseases, including pulmonary tuberculosis, bronchiectasis, rheumatic fever, recurrent furunculosis and infectious arthritis, were observed in 10 others. Adenocarcinoma of the rectum with metastasis to the liver was associated in 1 case. Twelve patients had distinct foci of infection (diseased tonsils, dental sepsis or prostatitis), but the degree of activity of the foci as septic atria or their relevance to the disease in question can be conjectured only on clinical grounds.

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The onset of dermatomyositis may be heralded by muscular, cutaneous or vasomotor signs. The commonest early complaint was of muscular pain and weakness in 18 cases, whereas in 14 cases edema and cutaneous lesions ushered in the disease. It is likely that in cases in which the onset is associated with a dermatosis subclinical muscular inflammatory changes are already present. In 6 more cases muscular and dermatologic symptoms were initially combined, in the form of eruption (or edema) and weakness, with or without pain. Vasomotor phenomena were observed in 2 cases before symptoms referable to the skin or to the musculature appeared.

Muscular Involvement.—There is no regularity or constant sequence for the distribution of the lesions of muscles. In 15 cases muscular symptoms were generalized from the start; in 24 the extremities suffered first, and in 1 case the lumbar muscles were first attacked. Usually the myositis was characterized by a nearly symmetric bilateral involvement. After establishment of the disease, the vulnerability of the muscles of the shoulder girdle, the arms and the neck was outstanding (figs. 1 and 2). In lesser degree the same was true of the gluteal and femoral muscles. Our material confirmed the often repeated observation that, in general, the distal musculature of the limbs suffers relatively less than the proximal. The muscles were usually spontaneously painful and tender on pressure, but in 6 cases pain was either inconspicuous or absent throughout the course of the disease. It is an interesting circumstance that extreme pathologic changes sometimes can be demonstrated in muscles which are not painful in the slightest degree on movement or on palpation. The consistency of the muscles may be normal, soft and doughy, or tough, firm and fibrous. Muscular atrophy usually developed in cases of longer duration, and dermatomyositis was frequently associated with muscular contractures and "fixed joints." The deep reflexes were occasionally normal in spite of muscular weakness, but more often they were decreased or absent.

Implication of the vital striated musculature (that of the pharynx and the larynx, the intercostal muscles and the diaphragm) is an extremely serious complication of dermatomyositis. As functional evidence of damage to muscles becomes more obvious, the hazard of fatal bronchopneumonia increases apace. Dysphagia, a consequence of myositis of the palatal muscles and pharyngeal constrictors, was a symptom of 27 patients. Thirteen patients demonstrated vocal changes, consisting of hoarseness or altered pitch. Ten patients experienced frank dyspnea, and the vital capacity of 2 of them was found to be less than half the normal volume.

Facial weakness often was noted, although paresis of the muscles of expression associated with dermatomyositis is not so startling as that

associated with myasthenia gravis. Weakness and tremor of the tongue occurred in 6 cases. Diplopia was complained of by 4 patients; this signified involvement of the extraocular group of muscles by the disease. Four patients presented transitory symptoms of sphincteric weakness;

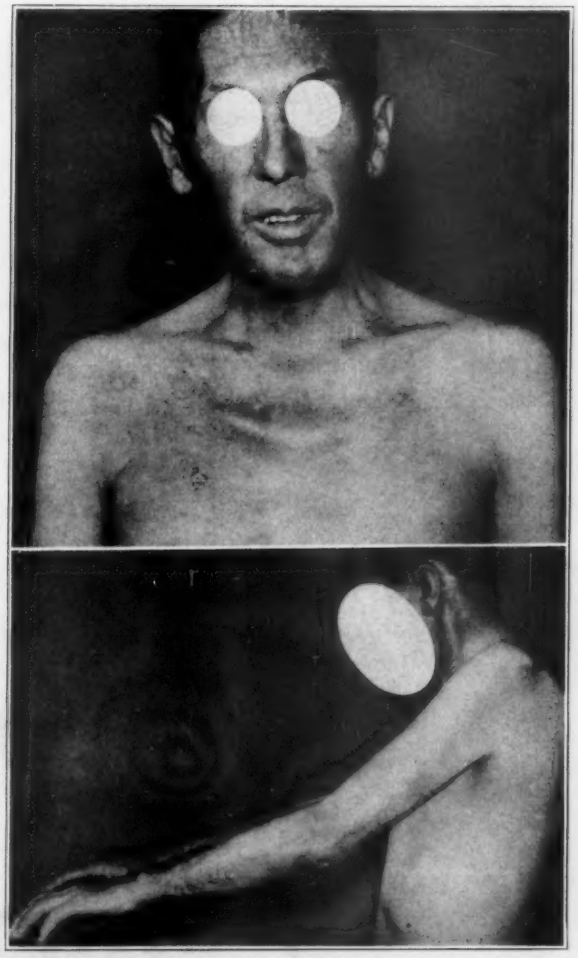

Fig. 1.—Different views of same patient with dermatomyositis of nine months' duration. The patient, aged 45, has muscular atrophy and weakness of shoulders and arms, mottled pigmentation over the anterior surface of the thorax and sclerosis of the skin.

in 3 cases there were symptoms referable to the anus, and in 1, symptoms referable to the vesical neck.

Cutaneous Manifestations.—Changes in the skin are of a diverse character and account largely for the variability of the clinical picture.

In 2 cases of our series cutaneous abnormalities were entirely wanting; yet, in such an event, the term "dermatomyositis" is still retained (although "polymyositis" may describe the syndrome more accurately) with the same propriety that obtains for the designation "pellagra" when that disease exists without cutaneous lesions.

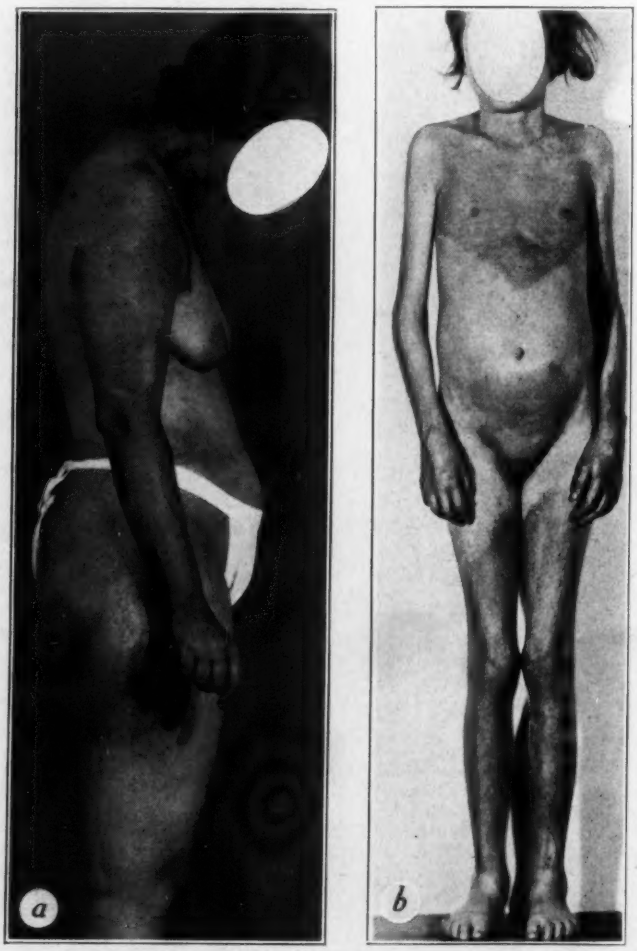

Fig. 2.—The first patient (*a*), aged 38, has had dermatomyositis for one year. There are slight sclerodermic changes in the skin of hands and forearms and extensive hyperpigmentation simulating that of Addison's disease. The weak and slightly atrophic musculature of the neck has caused the abnormal posture. The next picture (*b*) was taken of a 10 year old child one year after onset of symptoms. The erythematous eruption has features of disseminate lupus erythematosus. Wasting of the arms and thighs, as well as edema of the ankles, is evident.

Edema occurred frequently, alone or combined with a superficial exanthem, as a firm, diffuse and, at times, generalized process. The edema might pit somewhat on pressure or might be brawny, and ordinarily it was limited to the face and extremities. Erythema, if associated, was rosy or livid and diffuse or mottled and could be observed overlying edematous areas or involving skin otherwise unaltered. The distribution of cutaneous symptoms did not seem to be determined or affected particularly by the extent of involvement of the muscles. Periorbital edema, sometimes with erythema, independent of or associated with facial edema, was encountered in 25 cases, in nearly a third of which the swelling appeared concurrently with initial symptoms.

Mild pigmentary and atrophic changes sometimes accompanied the erythema, as sharply demarcated, patchy involvement of the dorsa of the hands and knuckles, the elbows and knees, or the face, neck and manubrial regions. The resulting lesions then showed certain resemblances to the dermatitis of pellagra. In a few instances, features similar to those of localized poikiloderma atrophicans vasculare were observed. Several times the predominant distribution of the cutaneous lesions over exposed parts was highly suggestive of some mechanism involving sensitivity to light.

In 4 patients the cutaneous changes consisted only of increased pigmentation of the face, neck, extremities and parts of the trunk. Five additional patients had hyperpigmentation in combination with what looked like mild scleroderma, although the pigmentation was not limited to sclerodermatous regions (fig. 2*a*). Seven other patients had a moderate degree of scleroderma or changes resembling those of scleredema adultorum, with induration and thickening of the affected regions but without pigmentary manifestations.

Our material included 3 cases in which the cutaneous picture was similar to that of disseminate lupus erythematosus (fig. 2*b*). In spite of close clinical parallelisms, however, the microscopic examination revealed characteristics that were distinguished readily from those of lupus erythematosus.[1] The picture of poikiloderma atrophicans vasculare (poikilodermatomyositis[2]) was recognized in a boy aged 6 years. O'Leary[3] expressed previously the concept that poikiloderma atrophicans vasculare may develop as a result of damage to the cutaneous vascular structure by various noxae and that it should be properly regarded, then,

1. Montgomery, H.: Pathology of Lupus Erythematosus, J. Invest. Dermat. **2**:343-359, 1939.

2. Petges; Mougneau; Lecoulant, and Delas: Poïkilodermie et polymyosite (poïkilodermato-myosite), Bull. Soc. franç. de dermat. et syph. **36**:817-823, 1929.

3. O'Leary, P. A., in discussion on Ebert, M. H., and Slepyan, A.: Lymphoblastoma, Arch. Dermat. & Syph. **39**:581-583 (March) 1939.

as a cutaneous response to various pathogenic agents rather than as an etiologically unique disease.

There were miscellaneous additional observations. Diffuse alopecia appeared on the scalp in 7 cases synchronously with early symptoms of the disease or within several months of its onset. Subcutaneous calcification supervened in 5 cases. One patient was shown roentgenologically to have extensive calcareous deposits in the soft tissues of the pelvic girdle and lower extremities; 4 of these 5 patients had stony nodules in localized zones, such as on the fingers or near the larger joints. Osteoporosis was demonstrated in the long bones of 5 of 6 patients subjected to roentgenologic examination. This demineralization possibly may be explained by the muscular weakness and inactivity of the extremities.[4] Stomatitis was found in 9 cases, the changes consisting of redness, edema and, sometimes, crusted erosions of the tongue, gums and buccal mucous membrane.

General Observations.—Fever, when present, was of a moderate grade and followed a remittent pattern. In only 11 cases was fever of any importance noted, and in these 11 except as a terminal manifestation the temperature remained lower than 102 F. (38.9 C.). Slight splenomegaly was detected in 8 cases as a firm, insensitive swelling. Mild enlargement of accessible lymph nodes was observed in 3 cases. In 5 the liver was increased in size. A history of a loss of 10 to 50 pounds (4.5 to 22.7 Kg.) since the onset of symptoms was furnished by about a third of our patients.

The pulse, in general, was accelerated proportionately to the fever or in excess of it. That the systolic blood pressure often tends to decrease is an impression gained from inspection of our data, and the fall may amount to as much as 40 mm. of mercury.

In 9 cases some objective evidence of cardiac abnormality was present. The heart was enlarged to a moderate degree in 5. Rheumatic disease was observed in 2 cases. In 4 cases electrocardiographic abnormalities were recorded, such as slurring of the QRS complex in leads I and II, diminished amplitude of the QRS complex or, in 1 case, inversion of the T wave in lead II. Vasomotor abnormalities (acrocyanosis and Raynaud's syndrome) were exhibited by 10 patients. Paresthesias occurred in 9 cases, as numbness or tingling of the fingers or generalized formication, but at no time were the symptoms pronounced, and usually they were inconstant. Profuse sweating, although frequently cited in the literature as a common accompaniment of dermatomyositis, was an inconsequential symptom in the cases observed at the clinic.

4. Schmidt, M. B.: Atrophie und Hypertrophie des Knochens einschliesslich der Osteosklerose, in Henke, F., and Lubarsch, O.: Handbuch der speziellen pathologischen Anatomie und Histologie, Berlin, Julius Springer, 1937, vol. 9, pt. 3, pp. 14-17.

Recently Bruce [5] described a complicating retinitis which paralleled the course of the muscle disease. That this must be an unusual event, however, is indicated by the absence of similar ophthalmoscopic findings in 30 of our patients who were examined.

Articular symptoms were recorded in 6 cases. It must be considered, however, that by certain subjects muscle pain will be referred to the region of the joints (tendinous insertions). Also, impaired mobility due to muscular contractures may be adduced erroneously by the patient as further evidence of articular disease. In 2 cases arthritis was present, whereas in the others the articular complaint consisted of transient pains appearing with initiation of the illness.

Laboratory Tests.—In spite of the subacute and chronic debilitating process which characterized the cases, anemia was not a frequent finding. At no time was the total number of erythrocytes less than 3,500,000 per cubic millimeter, nor was the concentration of hemoglobin less than 9.7 Gm. per hundred cubic centimeters. The number of leukocytes in most instances was not greater than normal. In only 5 cases did the number of leukocytes exceed 10,500 per cubic millimeter, and in only 8 cases did less than 5,000 per cubic millimeter prevail. Increase in the number of monocytes, to which attention has been directed by Moser,[6] was also prominent in our material. Of the 32 cases in which differential studies of the blood were made, in 13 there was a monocyte count of 8 per cent or more; a monocyte count of 17 per cent was the highest figure attained. In 26 cases the proportion of lymphocytes exceeded 20 per cent and averaged about 27 per cent. An eosinophil count to the extent of 5 per cent or more occurred in 11 cases, the highest value being 16 per cent. A relation between eosinophilia and duration or severity of the disease could not be ascertained. In 9 determinations an increased rate of sedimentation of erythrocytes was demonstrated, varying from 41 to 75 mm. per hour.

In 2 of 4 cases the level of serum protein was reduced to approximately 5.5 Gm. per hundred cubic centimeters. In 1 of these cases there was slight edema; in the other, moderate edema. Decrease of the albumin-globulin ratio was not found. Albumin, grade 1 to 2 (on a basis of 1 to 4), was present in the urine of 14 patients. Small numbers of hyaline or granular casts were associated occasionally in 5 of these cases.

Multiple determinations of urinary creatine were made in 6 cases, and the amounts excreted ranged between 250 mg. and 1,100 mg. in twenty-four hours. With this limited group the creatinuria was found generally

5. Bruce, G. M.: Retinitis in Dermatomyositis, Tr. Am. Ophth. Soc. **36:** 282-297, 1938.

6. Moser, K.: Myalgie und Myositis, in Bumke, O., and Foerster, O.: Handbuch der Neurologie, Berlin, Julius Springer, 1935, vol. 9, pt. 1, pp. 5-15.

to be greater when the condition was comparatively severe; whether further experience will support this observation remains to be seen. The results signify a metabolic error whereby the ability of the muscular tissue to bind, store, retain or utilize creatine is impaired.[7] Studies of elimination of porphyrin in urine and feces were conducted in 3 cases, with normal findings for practically all specimens examined.[8] In 2 cases adequate quantities of urinary ascorbic acid were excreted.

Examination of the cerebrospinal fluid in 9 cases revealed no abnormality.

ETIOLOGIC CONSIDERATIONS

In the material examined, the sexes were nearly equally represented, 18 males and 22 females. No age is exempt, but more patients in the fifth decade of life than in any other age period were included in this series. At the onset of the disease the youngest patient was 4 years old and the oldest 65. Of 34 cases in which data are available, the onset was more common (16) in the summer than in any other season, and in the majority of cases (23) the onset was in the summer and fall. There is evidence, already cited, that in many instances the level of general health was depressed prior to the onset of symptoms.

Since little is known of the exciting agent of dermatomyositis, many etiologic hypotheses have been advanced from time to time, too often based on observations in single cases. These will not be enumerated here, because they have been reviewed repeatedly by other authors. However, we shall report the results of certain bacteriologic examinations employed in the study of our cases, and we shall comment briefly on their significance.

Bacteriologic Studies.—There appears to be little doubt that dermatomyositis is directly or indirectly of infective origin, and evidence in the literature has incriminated various micro-organisms. Whether bacteria affect the muscles directly by dissemination by way of the blood stream or by means of toxic products which they elaborate must remain an open question. The first possibility implies a bacteremia, but in the transitory and passive sense of a migration to the muscles rather than of a frank infection of the blood stream (septicopyemia). A number of observers have found bacteria in the tissues either by cultural or by histologic methods. For the most part, streptococci and staphylococci have been demonstrated, although it must be stated that the authenticity of the clinical diagnosis in some of the cases is dubious.

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7. Steinitz, H., and Steinfeld, F.: Untersuchungen zur Kreatinstoffwechsel bei Dermatomyositis, Ztschr. f. d. ges. exper. Med. **79**:319-328, 1931.

8. Brunsting, L. A.; Brugsch, J. T., and O'Leary, P. A.: Quantitative Investigation of Porphyrin Metabolism in Diseases of the Skin, Arch. Dermat. & Syph. **39**:294-307 (Feb.) 1939.

In our series, cultures were made of 17 specimens of muscle taken for biopsy; most of the cultures were made by Dr. E. C. Rosenow or under his direction. In 7 instances organisms were not recovered. Streptococci were isolated from 5 of the specimens, once under anaerobic conditions. Animals were inoculated each time; in 3 instances hemorrhagic lesions of the skin and muscles resulted, which were interpreted as an elective localization of "myotropic" micro-organisms. Diphtheroids were cultured in 3 cases, associated once with diplococci and once with micrococci. The importance of diphtheroids presumably rests in the possibility that they may represent dissociated innocuous forms of streptococci.[9] Barker,[10] however, has expressed doubt concerning their etiologic significance. Of the 3 remaining cases, staphylococci and diplococci were observed in 1, small gram-positive micrococci in the second and gram-negative bacilli in the third. Unfortunately, virulence tests were not performed with the nonstreptococcic cultures on animals. Bacteria were not demonstrated in tissues at any time by staining with a modification of Gram's method. From the nasopharyngeal swabbings of 6 of the patients, streptococci were recovered which when injected intravenously into rabbits produced multiple hemorrhagic muscular lesions.

We believe it prudent to adopt a conservative stand regarding the implications of the aforementioned bacteriologic results. Until repeated confirmatory observations are available, judgment of the exact etiologic significance of the bacteria should be reserved, and we concur in the opinion of von Zalka [11] that one cannot maintain, because bacteria are demonstrable intramuscularly in certain cases, that the condition in every case is due to the local activity of bacteria. For the present it is best to accept the broad hypothesis that the manifestations of dermatomyositis may be caused by various bacterial factors, either as a direct invasion by way of the blood stream or as a toxic effect of bacterial products on the reacting tissues.

PATHOLOGIC CHARACTERISTICS

The following account of histologic alterations in the skin and musculature due to dermatomyositis is based on a survey of material obtained for microscopic study, comprising 20 specimens of skin and 23 specimens of muscle excised from 28 patients. In addition, the protocols and

9. Koch, R., and Mellon, R. R.: The Biological and Clinical Significance of Diphtheroids in the Blood Stream, J. Bact. **19**:25-27, 1930.

10. Barker, N. W.: An Unusual Case of Dermatomyositis, Proc. Staff Meet., Mayo Clin. **5**:169-172, 1930.

11. von Zalka, E.: Ueber einen seltsamen Fall von Polymyositis, Virchows Arch. f. path. Anat. **281**:114-128, 1931.

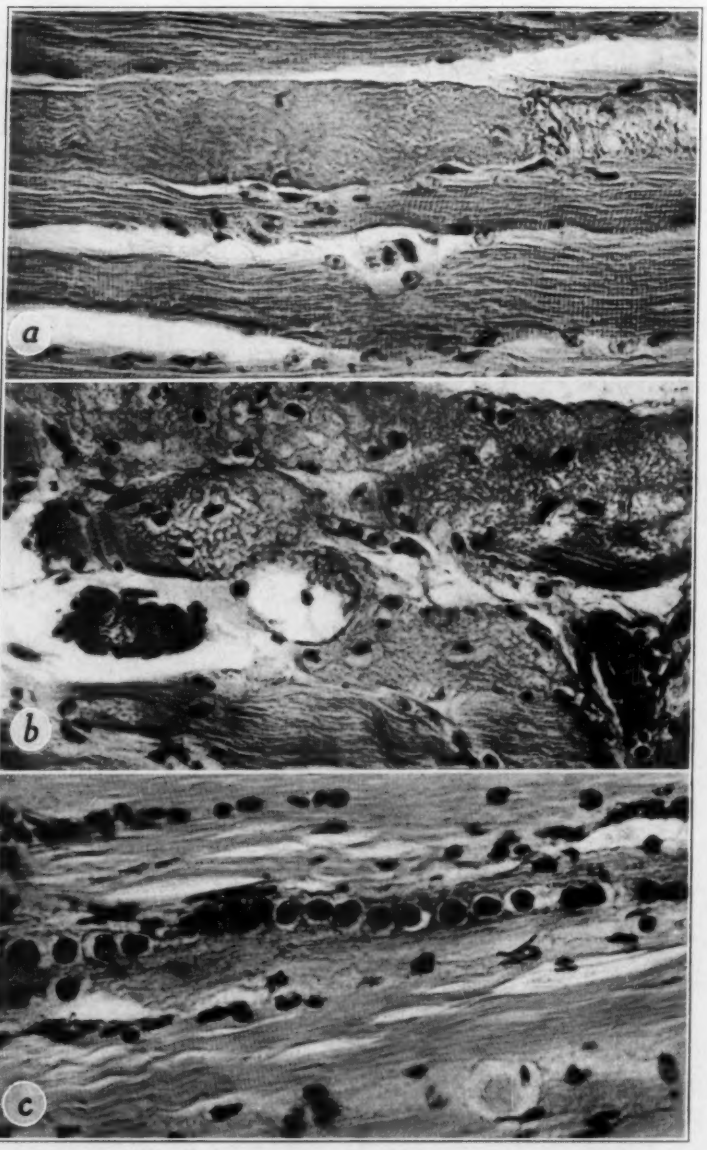

Fig. 3.—Photomicrograph *a* is a section of muscle from a case in which the condition was of one month's duration. The bundles are of irregular and unequal thickness; granular parenchymal changes and slight interstitial edema are present, but inflammatory infiltrate is lacking (× 375). Photomicrograph *b* shows granular, fibrinous and vacuolar degenerative changes in muscle. Cross striations are preserved in limited portions of the damaged fibers (× 350). Photomicrograph *c* shows nuclear multiplication resulting in long axial files of nuclei. This has been regarded as a sign of regeneration (× 375).

histologic preparations of 4 necropsies have been available; in 3 of these cases, specimens of tissue also were secured for biopsy prior to the patient's death.

Histologic Alterations of the Muscle.—The salient microscopic features in the muscle comprise parenchymal and interstitial changes of nonspecific character and varying degree. It is our impression that the initial pathologic reaction is revealed within the bundles of muscles themselves [12] (fig. 3 *a*). These exhibit proliferation of nuclei, variability in diameter, effacement of transverse striations, separation of the myofibrils and occasionally coagulation or hyalinization of the sarcoplasm. There is evidence in our material that interstitial inflammation occurs secondarily, possibly as a response to the parenchymal damage. In addition to the changes already enumerated, there occur fragmentation of fibers, granular, fibrinous and vacuolar degeneration (fig. 3 *b*), basophilic metachromasia, and phagocytosis by large histiocytes. The nuclei of muscle cells are often abundantly increased in number (fig. 3 *c*). Often the cellular infiltrate attains only meager proportions even when severe and extensive myositis exists, another indication that inflammatory phenomena are not the essential ones in lesions of dermatomyositis (fig. 4 *a*). Dermatomyositis of long standing is associated with more severe and widespread destruction of the muscle bundles. Interstitial infiltrate and edema may be slight or outstanding. Cellular collections, largely of lymphocytes, histiocytes, plasma cells and fibroblasts, are observed either accumulated perivascularly or invading diffusely. The blood vessels are dilated and engorged, but as a rule the walls reveal no remarkable abnormality. At times replacement by fibrosis supervenes prominently in myositis of long duration. The diaphragm and the pharyngeal and ocular muscles show changes identical with those appearing in the skeletal musculature (fig. 4 *b* and *c*).

Myocarditis was demonstrated in 2 cases at necropsy. In 1 the inflammation was associated with subacute mitral endocarditis and obliterative pericarditis; yet, because Aschoff nodules were not found on diligent search, the possibility of independent myositis of the cardiac muscle could not be excluded. In the second case pronounced similarity of histologic changes was observed in the cardiac and skeletal muscles. Over extensive regions the bundles appeared swollen, and the sarcoplasm hyalinized or revealing a grayish tinctorial reaction. The cross striations were obscured, and many nuclei stained poorly. A mild scattering of lymphocytes and histiocytes occupied the interstitial tissue.

Histologic Changes of the Skin.—In general, the cutaneous changes in any phase of the disease are of a banal inflammatory character. The

12. Waisman, M.: Dermatomyositis: A Clinical and Histopathologic Study of Forty Cases, Thesis, University of Minnesota Graduate School, 1939.

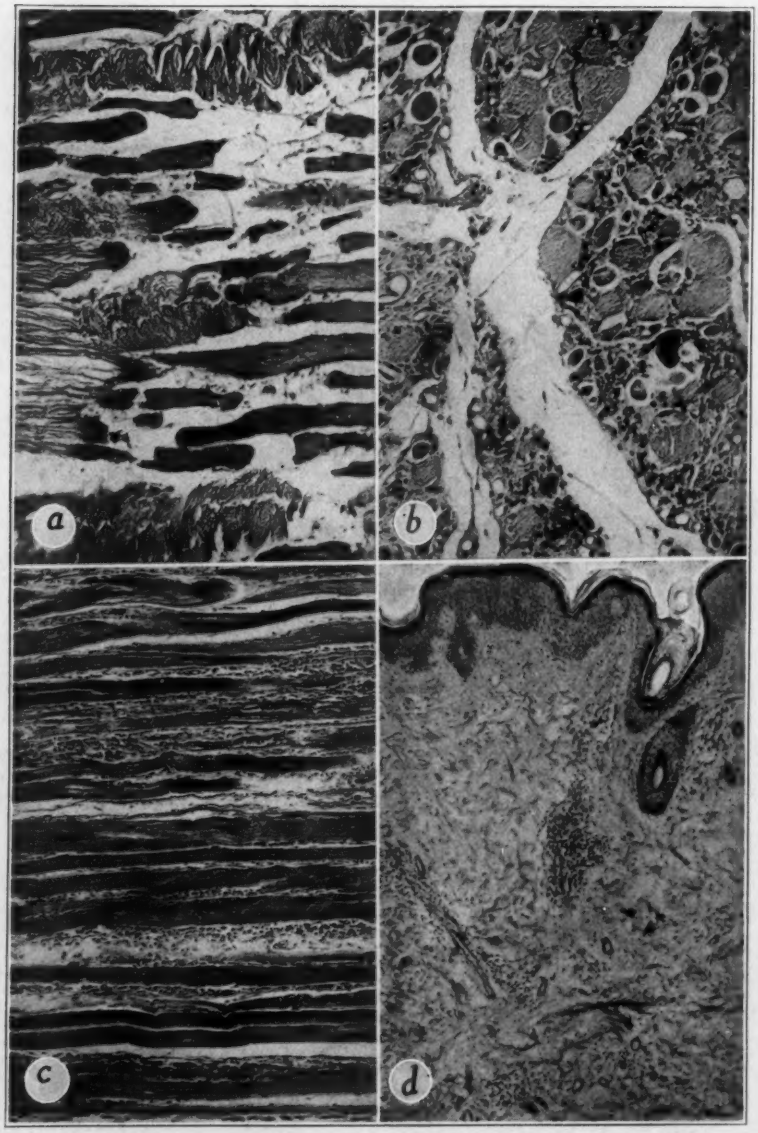

Fig. 4.—Photomicrograph *a* shows striking degenerative alterations in the sarcoplasm, without interstitial inflammation (× 100); a cross section (*b*) through superior constrictor muscle of the pharynx shows variability in diameter of fibers, homogenization of isolated fibers and inflammatory infiltrate invading the endomysium (× 100). Diaphragmatic myositis is shown (*c*), with dense interstitial invasion by inflammatory cells (× 100). The skin (*d*) of the patient illustrated in figure 2 *b* shows follicular keratotic plugging, mild edema and perivascular lymphocytic infiltrate. The picture is one of toxic dermatitis and would not be confused with that of lupus erythematosus (× 40).

epidermis may be the site either of mild acanthosis or of atrophy. The collagen in the cutis is often normal, but at times the fibers appear to be swollen, pale, granular, opaque or homogenized. In some cases mild edema separates the fibers, either generally throughout the cutis or only in the upper or lower portions. Perivascular cellular collections seem to be a constant feature, composed largely of lymphocytes, often with small numbers of plasma cells and histiocytes and sometimes fibroblasts and mast cells, all suspended on a delicate reticulum. The infiltrate, which may be of mild or medium degree, is limited usually to the upper and the middle portion of the cutis, but in a few cases it may be present only in the lower zone. Often the inflammatory cells surround dermal appendages. Occasionally a mild diffuse or perivascular infiltration of lymphocytes and large mononucleated cells occurs just beneath the cutis, but in none of our sections was the adipose tissue involved in its entire thickness.

From patients clinically showing scleroderma-like infiltration 8 specimens of skin were available for biopsy. Only 2 of these showed histologic features indicative of true scleroderma, such as homogenization and condensation of connective tissue and slight thickening of a few vascular walls. In 4 of the cases there were observed some homogeneous staining of collagen and some edema separating the bundles of collagen, a structure recalling, in some respects, that of scleredema adultorum. One other specimen displayed an extensive perivascular inflammatory reaction, particularly in the lower cutis, with encroachment on the upper zones of the subcutis. In the remaining case important microscopic abnormalities were not detected. These observations apparently indicate that the characteristic cutaneous histologic changes associated with scleroderma are not commonly encountered in lesions of dermatomyositis. Induration, which suggests scleroderma clinically and which even may persist indefinitely, is probably most often due to edema and sometimes to inflammatory infiltration. The morphologic similarity of dermatomyositis and scleredema adultorum is of interest; Gottron [13] and Grzybowski [14] have been led, by the demonstration of a common metachromatic substance infiltrating the skin in cases of scleredema [15] and in certain instances of dermatomyositis, to postulate a relation between the two diseases.

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13. Gottron, H.: Hautveränderungen bei Dermatomyositis, in Comptes rendus des séances, VIII[e] Congrès international de dermatologie et de syphiligraphie, Copenhagen, Engelsen and Schrøder, 1930, pp. 826-830.

14. Grzybowski, M.: Zur Kenntnis der Dermatomyositis, Arch. f. Dermat. u. Syph. **174**:541-552, 1936.

15. Freund, H.: Ueber Sklerödem (Buschke), Arch. f. Dermat. u. Syph. **161**:92-113, 1930.

The microscopic picture disclosed in 2 cases in which the condition clinically simulated lupus erythematosus was that of "toxic dermatitis," a nonspecific inflammatory process (fig. 4 *d*). Biopsy was not performed in our case of poikilodermatomyositis. Dowling and Freudenthal [16] recently have concluded, after a study of the problem, that the skin in cases of poikilodermatomyositis is not the same histologically as that in true cases of poikiloderma atrophicans vasculare.

COURSE AND PROGNOSIS

As a rule the tempo of the onset predicates the rapidity of the course that will follow. Dermatomyositis with acute onset surges quickly forward to a severe grade of involvement; when the condition begins slowly it is more likely to lead to milder illness and disability and a favorable outcome. There are cases, however, in which the condition begins acutely but pursues a mild course; others in which there are slight or severe exacerbations with intervals of improvement, and still others in which the onset is gradual but the condition seems to accelerate in intensity and ends fatally, with an overwhelming seizure of the whole striated musculature.

A single relatively brief remission characterized the course of the disease in 6 of our cases, the duration of improvement commonly not exceeding several months. As a generalization, it may be stated that usually the course is progressive up to a certain point, after which, if the patient survives, activity of the disease ceases and repair tends to set in.[17] Of 38 cases which were followed, in 19 death was known to have occurred; in this group four months was the shortest extent of the disease, and six years the longest, with an average duration of about two years. The cause of death was ascertained in 10 cases: 6 patients died of bronchopneumonia; 3, of respiratory paralysis, and 1, of cardiac failure. With the group of patients analyzed according to extremes of age, 1 of the 7 patients who were less than 15 years of age died, and 2 of the 5 who were more than 50 years of age died.

So far as we know, 4 patients have recovered completely. They were seen at the clinic between 1926 and 1933, so that an adequate interval has elapsed to rule out transitory remission. In none of these was the dermatomyositis severe. In 7 of the remaining patients there is improvement or the condition has reached a stage that is stationary (in

16. Dowling, G. B., and Freudenthal, W.: Dermatomyositis and Poikilodermia Atrophicans Vascularis: Clinical and Histological Comparisons, Brit. J. Dermat. **50**:519-539, 1938.

17. Weber, F. P.: Sequel to a Case of Polydermatomyositis, Proc. Roy. Soc. Med. **27**:138, 1933.

some, possibly, a healed stage), although accompanied by a variable amount of muscular incapacity. In only 2 of these 7 patients have the cutaneous changes largely disappeared; residual lesions encountered among the others include sclerodermic thickening, calcification in the subcutis and pigmentation.

Of the 8 patients whose condition has not improved, 2 have been too recently under initial observation to permit comment on the course of the disease. Three others lately have suffered relapse after a previous interlude of improvement that lasted less than six months. The remaining 3 have reported a slow, steady increase of weakness.

DIFFERENTIAL DIAGNOSIS

In spite of the variability of individual characteristics of dermatomyositis, as recounted in the foregoing paragraphs, any particular case usually presents a sufficiently suggestive ensemble at least to arouse the clinical suspicion of the observer. In the case of advanced disease, when classic features prevail the diagnosis as a rule is not formidable. Diagnostic problems are provided mostly by the early forms, encountered clinically before distinctive muscular and cutaneous signs have developed.

Most difficult to distinguish from dermatomyositis in certain instances in the early stages are diffuse scleroderma in its edematous stage and scleredema adultorum (Buschke). As Brock [18] has pointed out, even with the aid of histologic studies, it is often impossible to distinguish between early dermatomyositis and scleroderma, because changes in the two diseases may then be identical, both in muscle and in skin. The scleroderma-like metamorphosis which may occur later in the course of dermatomyositis is not true scleroderma. Indeed, one of the remarkable clinical qualities of this secondary cutaneous reaction is the relatively mild and limited sclerosis present, considering the duration of the disease. In well developed lesions, microscopic characteristics become more distinctive, as has been indicated. The changes in lesions of dermatomyositis generally tend to recede after attaining a certain intensity, whereas in those of primary scleroderma progression is the rule and the outlook is for increasing involvement. The arguments of Dowling and Griffiths [19] purporting to establish dermatomyositis and progressive scleroderma as the same disease are refuted by our studies as well as by those of Brock.[18]

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18. Brock, W. G.: Dermatomyositis and Diffuse Scleroderma: Differential Diagnosis and Reports of Cases, Arch. Dermat. & Syph. **30**:227-240 (Aug.) 1934.

19. Dowling, G. B., and Griffiths, W. J.: Dermatomyositis and Progressive Scleroderma, Lancet **1**:1424-1428, 1939.

Scleredema adultorum [20] appears in the wake of some acute infectious disease. Beginning with brawny swelling on the neck, it spreads upward over the face and downward over the arms and trunk within a few weeks. Often the lower extremities are not affected. The skin feels deeply indurated, and movements become greatly restricted, although they are usually painless. Occasionally the disease permeates to the subjacent muscles, making the distinction of it from dermatomyositis increasingly difficult. Scleredema nearly always reaches its height within a few weeks, remains stationary for a variable period and then undergoes complete or nearly complete involution in several months to three years.

Acute forms of dermatomyositis may require differentiation from trichinosis.[21] If the myositis is of epidemic character, dermatomyositis is immediately ruled out. An episode of gastroenteritis follows the ingestion of trichinous pork, affecting possibly several members of a family or many persons in a community. The muscles, predominantly of the extremities, become painful, tender and swollen. Characteristic symptoms may be caused by involvement of the cervical, intercostal, laryngeal or ocular muscles. Edema of the face appears in this early stage, localizing particularly around the eyes, but it disappears within a few days. The muscle pains reach their peak two or three weeks after infestation and then decline. In spite of the incidence of increased numbers of eosinophils in the blood in cases of dermatomyositis, a high degree of eosinophilia is more suggestive of trichinosis. The intradermal reaction of Bachman with trichinella protein antigen is a valuable aid in supporting the diagnosis.

In cases of acute and subacute disseminate lupus erythematosus,[22] the onset is often insidious; complaints of asthenia and disability sometimes precede the cutaneous signs by months. Pain in the muscles and joints and along the course of nerves may be severe. The symptoms, together with the weakness and eruption, may simulate closely those of dermatomyositis. Montgomery [23] recently has reemphasized the characteristic histologic changes in the skin in cases of lupus erythematosus. Study of striated muscle in 2 fatal cases of lupus erythematosus revealed no pathologic alterations. We believe, therefore, that data obtained at biopsy probably can be the means of distinguishing with accuracy between two

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20. O'Leary, P. A.; Waisman, M., and Harrison, M. W.: Scleredema Adultorum, Am. J. M. Sc., to be published.

21. Pepper, O. H. P.: Trichiniasis, in Christian, H. A.: Oxford Medicine, New York, Oxford University Press, 1936, vol. 5, pt. 3, 996 (1) and 996 (2).

22. O'Leary, P. A.: Disseminated Lupus Erythematosus, Minnesota Med. **17:** 637-644, 1934.

23. Montgomery, H.: Disseminate Lupus Erythematosus as a Systemic Disease, in Christian, H. A.: Oxford Medicine, New York, Oxford University Press. 1939, vol. 4, chap. I-C, pp. 44 (41)-44 (65).

diseases which clinically may be indistinguishable from one another at the time of observation.

Pellagra, on occasion, may be confused with dermatomyositis not only because the dermatitis may be similar but also because in cases of pellagra signs of asthenia and muscle tenderness may be prominent.[24] Polyneuritis, myasthenia gravis, the myopathies, panniculitis, purulent embolic (septic) myositis, erysipelas, Addison's disease, acute rheumatic fever, periarteritis nodosa, postinfectious asthenias and functional states of exhaustion may be simulated at times in the course of development of dermatomyositis.

TREATMENT

For this disease of unknown cause, treatment is comprised solely of empiric and symptomatic measures. General care of the patient and maintenance of a satisfactory nutritional state assume primary importance. Concentrates of vitamins should supplement the diet. When dysphagia is present, resort must be had to liquid feedings or even to feeding by means of a nasal tube. Respiratory infections are resolutely to be guarded against because of the hazard of complicating bronchopneumonia; for this reason residence in a dry, warm, equable climate is recommended. If feasible, foci of infection should be removed. Autogenous vaccines prepared from material from the nasopharynx, from septic foci or from muscles, when cultures have been satisfactory, have been administered in our cases but with varying degrees of encouragement. The use of aminoacetic acid has been disappointing in this disease, both alone and in combination with ephedrine. No significant influence on the strength of muscles was forthcoming in 3 cases from the use of prostigmine. Four cases in which sulfanilamide was given over what was considered an adequate period of time showed no beneficial results attributable to the drug. We believe, nevertheless, that sulfanilamide and its derivatives are deserving of further trial early in the course of the disease because of the otherwise hopeless prognosis in many of the cases.

Recently treatment with inhalations of oxygen has been instituted with the objective of eliminating the influence of anoxemia. The oxygenation of arterial blood is probably deficient in cases of dermatomyositis because of the lowered vital capacity. Subjectively, a beneficial effect is quickly appreciated when treatment with oxygen is carried out. The gas is administered economically and without discomfort for several hours or even throughout most of the day by means of the inhalation apparatus,[25] devised by Boothby, Lovelace and Bulbulian.

24. O'Leary, P. A.: Pellagra: A Study of Thirty-Four Cases in Localities Where Pellagra Is Not Endemic, Northwest Med. **27**:319-323, 1928.

25. Lovelace, W. R., II: Oxygen for Therapy and Aviation: An Apparatus for the Administration of Oxygen or Oxygen and Helium by Inhalation, Proc. Staff Meet., Mayo Clin. **13**:646-654, 1938.

Unless the patient is mortally ill, fever therapy is decidedly of value. Fever is induced by means of typhoid vaccine intravenously or by the Kettering hypertherm, pyrexia being kept well within the limits of the patient's tolerance. Inhalation of oxygen throughout the period of induced fever seems to aid the effects of the treatment materially.[26] In cases in which the condition is mild, stationary or arrested, physical therapeutic measures may further improvement by means of procedures which utilize penetrating warmth and gentle massage.

Other agents which were used occasionally and with doubtful or no benefit included thyroid, extract of adrenal cortex, insulin, quinine, potassium iodide, gentian violet (given intravenously), neoarsphenamine, sodium gold thiosulfate, and roentgen radiation applied to the muscles.

SUMMARY

In 40 cases of subacute and chronic dermatomyositis which were observed at the Mayo Clinic in the past thirteen years pertinent clinical, laboratory and histologic features have been noted. Analysis of the material in an attempt to elicit clues as to underlying etiologic factors indicates a high incidence of disease antecedent to the onset of dermatomyositis. Bacteriologic studies point to the possibility of localization of various micro-organisms in muscle. We agree with the hypothesis that dermatomyositis may be caused either by bacterial invasion from the blood stream or by toxic action of bacterial products on the muscular and cutaneous structures.

Symptoms may be inaugurated by muscular, cutaneous, musculocutaneous or vasomotor phenomena. Abnormalities occur in the consistency, volume, sensibility and function of muscles and often with serious effects when the vital striated musculature becomes involved. A remarkable feature of the disease is its diverse dermatologic manifestations. Occasionally cutaneous changes are absent, but in the preponderance of cases they are prominent and include erythema, edema, pigmentation, sclerosis, atrophy, lupus-erythematosus-like efflorescence and poikiloderma atrophicans vasculare. The systemic reaction is indicated in some cases by fever, which only terminally is high. Abnormal metabolism of creatine is probably a constant occurrence in cases of dermatomyositis.

The course is characterized usually by progression up to a certain point, after which recovery tends to set in. Muscular and cutaneous residua are the rule. Approximately half the patients in the group observed at the clinic have died.

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26. Krusen, F. H., and Elkins, E. C.: Fever Therapy by Physical Means, J. A. M. A. **112**:1689-1696 (April 29) 1939.

Microscopic study of the earliest lesions of muscles indicates that the primary reaction may be manifested by the sarcoplasm rather than by interstitial cellular infiltration. The skin usually discloses only nonspecific changes, particularly those of inflammation and edema.

Diseases which may require diagnostic differentiation from dermatomyositis are enumerated, and emphasis has been placed on the difficulties encountered in some cases of early stages of diffuse scleroderma and scleredema adultorum.

The methods of treatment which we have utilized are outlined and include the administration of vaccines, aminoacetic acid and sulfanilamide. It appears, at the present time, that therapeutic promise is offered by the suitable combination of artificial fever therapy, inhalations of oxygen and physical therapy.

ECZEMA OF THE EAR ASSOCIATED WITH A DIPHTHERIA-LIKE ORGANISM

REPORT OF A CASE

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AND
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The case herein reported is one of probable diphtheritic eczema of the skin of the auricle and external auditory canal. Bart [1] over one hundred years ago called attention to this infection. In 1922 Biberstein [2] made a classification of the disease and gave therapeutic suggestions.

REPORT OF CASE

The patient, a white woman aged 32, was first examined on Feb. 15, 1939, at which time she presented an erythematous vesicular and exudative dermatitis involving the right auditory canal, the entire right auricle and a large area of skin on the right side of the face and neck. There was a purulent discharge from the middle ear. She gave a history of infection of the middle ear which began four months previously. During the past month the middle ear was treated by an otologist. About this time the right auricle became reddened and edematous (fig. 1). Shortly afterward vesicles appeared, which ruptured and exuded a watery substance.

After our examination the patient was given fractional doses of roentgen rays and local applications of wet compresses and emollients. She improved slowly over a period of two months, after which she underwent an operation for the middle ear infection.

On October 20 the patient returned, with edema and a rather sharply bordered erythema of the right auricle and adjacent areas. Vesicles, crusts and exudation were present. There was no visible discharge from the middle ear, and there were no pseudomembranes. A microscopic examination was made, and bacilli having the characteristics of the diphtheria group were observed (fig. 2). The culture was reported positive for Corynebacterium diphtheriae by the diphtheria division of the New York city health department. The virulence test was reported negative.

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1. Bart, S.: An Inquiry into the Nature, Cause and Cure of the Angina with Suffocativa or Sorethroat Distemper, as It Is Commonly Called by the Inhabitants of This City and Colony, New York, S. Inslee and A. Car, 1771.

2. Biberstein, H.: Ueber Hautdiphterie insbesondere die ekzematoide Form, Med. Klin. **18**:168-174, 1922; Die Diphtherie der Haut, in Jadassohn, J.: Handbuch der Haut- und Geschlechtskrankheiten, Berlin, Julius Springer, 1939, vol. 9, pt. 1.

Fig. 1.—Patient with diphtheritic eczema after local treatment for twenty-four hours. The entire external ear was covered with an erythematovesicular eruption. No pseudomembrane was present.

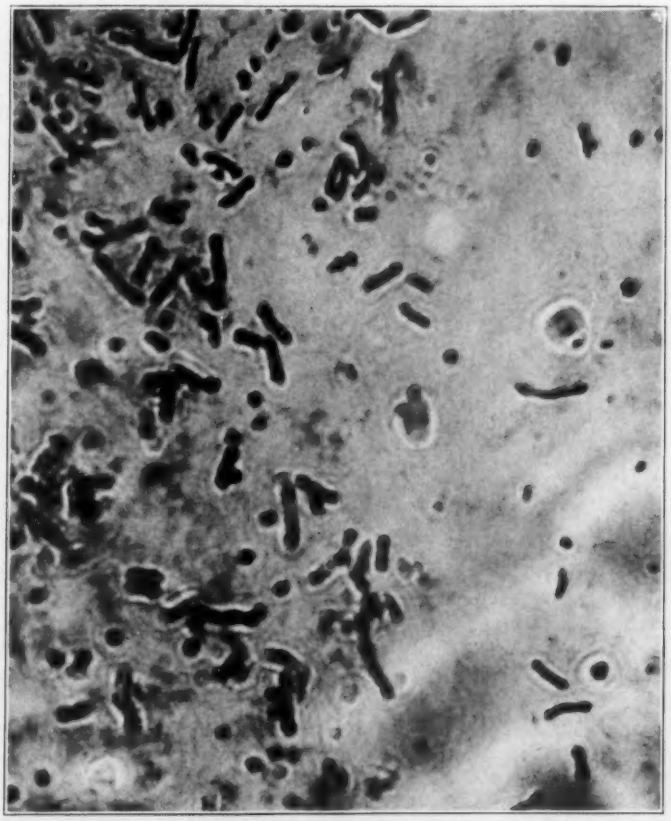

Fig. 2.—Photomicrograph of a smear taken from the ear, showing numerous club-shaped diphtheria-like bacilli in typical formation.

The following ointment was applied to the ear daily after the application of basic isoamylhydrocupreine (eucupin) 5 per cent in 50 per cent alcohol.

	Percentage
Rivanol [3]	1
Salicylic acid	3
Castor oil	5
Zinc oxide ointment to make	100

Within forty-eight hours the eruption had disappeared from all visible areas. The patient, who lived out of town, thought herself entirely well and discontinued treatment. A few days later a serous discharge was noticed coming from the auditory canal, and this was followed by a recurrence of the eruption on the auricle. The presence of diphtheria bacilli in the secretion of the canal was again observed. Because of the involvement deep in the external auditory canal, the patient was unable to reach these areas with local applications, and serum injections of diphtheria antitoxin were given in addition to the local treatment. According to the last report from the patient, she had improved.

This case is reported for the following reasons:

1. When eczema, especially in this location, does not respond to the usual treatment for simple eczema or infectious eczematoid dermatitis, the presence of diphtheria bacilli should be suspected.

2. When the presence of diphtheritic eczema has been proved microscopically, culturally and sometimes by virulence tests, the local treatment is usually sufficient to cause the disappearance of the eczema in two to four days. In occasional cases a longer time may be required.

3. In contrast to the success obtained by serum treatment in the deeper forms of diphtheria, the eczematoid form responds to local treatment as previously outlined more quickly, and the complications sometimes associated with administration of serum are eliminated. When the infection cannot be reached by local applications, injections of serum are necessary.

4. A positive reaction to a virulence test is of great aid in the diagnosis of diphtheria of the skin but is not entirely necessary, as the diphtheria bacilli have a varying virulence for different species and even for the same species. The degree of virulence even for the same strain may be variable, so that the virulence tests may only show the different degrees of virulence, but this test does not differentiate true diphtheria from pseudodiphtheria.

The clinical features, the presence of bacilli of the diphtheria group, proved microscopically and by culture, and the prompt local therapeutic response of accessible areas we believe confirm the diagnosis of diphtheritic eczema. However, some physicians believe that a positive reaction to a virulence test is necessary for a definite diagnosis of eczema due to the diphtheria bacillus.

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3. Rivanol is 2-ethoxy-6, 9-diaminoacridium hydrochloride.

SUPERFICIAL NONINFLAMMATORY LESIONS OF THE FEET

DEMONSTRATION IN SECTIONS OF A FUNGUS OF THE GENUS ACTINOMYCES

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LOS ANGELES

Ormsby and Mitchell,[1] in a report on 65 proved cases of ringworm of the feet in 1916, confirmed a previous classification made by Kaufmann-Wolf,[2] who divided his cases into three groups according to the clinical manifestations, viz., vesicular, squamous and pyodermic.

Herein are presented data regarding 2 cases in which lesions on the feet were believed to be occasioned by a fungus. They demonstrated certain clinical features in common which were not embraced by the aforementioned descriptions, no reference to which has been found in the literature. I recall observing a few cases with similar lesions in the past but attached no more importance to them than did the patients themselves, some of whom were unaware of their existence. It was presumed that these early lesions were probably dystrophies of superficial circumscribed areas of the stratum corneum due to pressure, as they appeared to be distributed over the areas of the soles where impact and following pressure would be greatest; furthermore, they were devoid of any signs of inflammation.

REPORT OF CASES

CASE 1.—A white youth aged 17 presented himself in June 1936 complaining of "warts on the feet." He first noticed the lesions about two weeks previously, while cutting his toe nails. He was unaware how long they had existed; he had never seen anything like them before, and no other member of the family was similarly affected. The lesions did not cause subjective symptoms at any time. He had visited a chiropodist, who informed him that they were warts.

The lesions as presented can best be described as simple losses in continuity of small rounded areas of the superficial layer of the skin of the soles. They appeared to be distributed over areas subjected to greatest pressure, viz., toes, anterior arch and ball of heel. During their early development they appeared as small

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Read in part before the Symposium Society.

1. Ormsby, O. S., and Mitchell, J. H.: Ringworm of Hands and Feet, J. A. M. A. **67**:711 (Sept. 2) 1916.

2. Kaufmann-Wolf, M.: Ueber Pilzerkrankungen der Hände und Füsse, Dermat. Ztschr. **21**:385, 1914.

rounded entities and somewhat suggested plantar warts, but the corneous collarette and "seeds" usually associated with plantar warts were absent. The affected areas slowly become polycyclic in outline, owing to the coalescence of adjoining lesions; while under the influence of a fungicide they gradually became shallower, until finally normal skin resulted. No other lesions were present during the time of observation. The webs of the toes in particular were subjected to careful inspection for macerated areas at each visit over a period of ten weeks. During this time the feet were checked with regard to perspiring; the findings supported the observation previously made by the patient that his feet were "always dry."

A series of scrapings were examined microscopically, with negative results. Plantings of the same material on Sabouraud's medium and dextrose agar rendered only colonies of cocci. This procedure was repeated at intervals for about three weeks, with similar results, during which time no medication was employed. In spite of the several failures it was felt that some information might be obtained from an examination of a section. Unfortunately, the patient was most averse

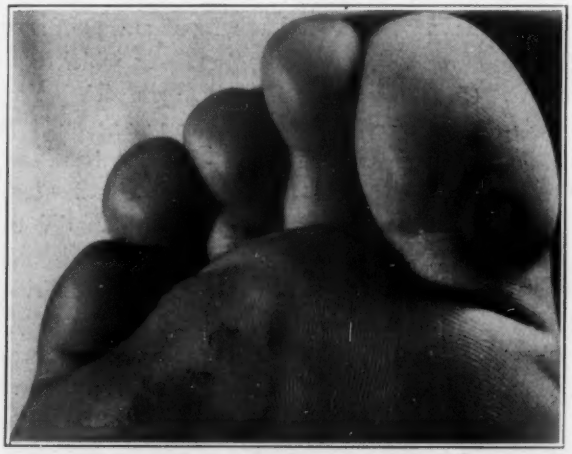

Fig. 1 (case 1).—Clinical appearance.

to having a biopsy specimen taken from the under surface of his foot and consented only when he was assured that it would not go below the more superficial layers of the skin. This was agreed to of necessity and because it was believed that as the process was clinically a noninflammatory one the morbidity might be contained and manifested in such a superficial section.

A segment was taken through a small rounded lesion situated anterior to the groups of lesions on the ball of the heel where the thickest skin was. It was sufficiently deep to cause a slight flow of blood. The section, however, demonstrated that no rete malpighii was removed. The stained sections showed what was apparently a normal corneous layer, with a loss of the superficial layers of keratotic cells over the affected part, so that the tissue demonstrated a small shallow depression on its surface. Immediately beneath the surface of the depression and lateral to it was noted a differentiation in the cells of the horny layer; they took the stain well along a line adjacent to the indentation, seemingly walling it off. Within the depression was found a narrow translucent to opaque band lining its base, which contained numerous small spores and fine mycelial

filaments. The latter ramified down through the base of the lesion, penetrating into the more densely stained layer of cells, beyond which they did not extend, as careful search beneath this layer failed to reveal their presence.

It was presumed that the negative results of cultures of the scrapings might have been occasioned by the fact that the scrapings were taken too superficially, as the section showed that the contained mass did not extend to the surface skin adjoining the erosion. Later scrapings taken from the base of these lesions and planted on Sabouraud's medium gave negative results. However, this was anticipated, as the patient had been given a fungicide to use after the removal of the

Fig. 2 (case 2).—Clinical appearance.

biopsy specimen. Further work was not possible, as the patient left to attend an eastern school, from which he notified me about two months later that the lesions had entirely disappeared. I had an opportunity to examine his feet on April 10, 1939, and found the skin to be perfectly normal.

CASE 2.—An actor aged approximately 50 complained of having "scalded areas" on his feet. They occasioned no distress. He said that he had noted them first while attending to his toe nails. The term scalded was apt, as the lateral and posterior aspects of the heel had the appearance of having been scalded, due to the fact that many of the lesions had coalesced to form a shallow pale purplish band. The lesions on the sole proper, however, were the typical shallow rounded

entities previously described. Scrapings from the base of one of these lesions when stained demonstrated fragments of mycelium of fine caliber. The patient did not permit a biopsy specimen to be taken. Several cultures were made from scrapings taken from the bases of many of the lesions. These gave negative results

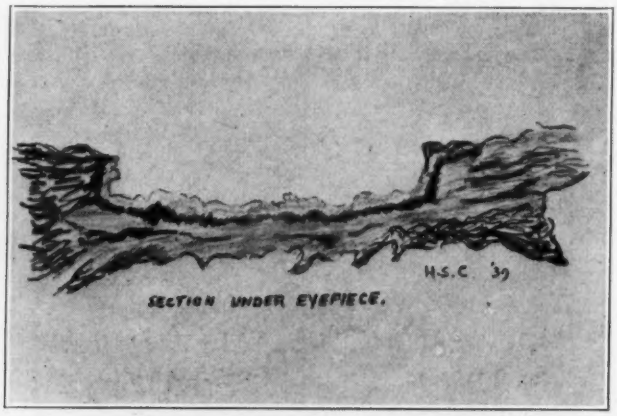

Fig. 3.—Ocular view of section.

Fig. 4.—Angle of the depression under low power.

with the exception of two tubes, which showed somewhat similar slowly growing anaerobic entities, which died out after transplantation.

The mycelium as manifested in the section was not comparable in size with any of the more common fungous entities found in infections of the feet. Its restricted distribution was also somewhat foreign, while the absence of all signs of inflammation seemed to place the condition in a category as interesting as it was unique.

Both Dr. Weidman and Dr. Plunkett, after examining the section independently, expressed their tentative opinions that the organism belonged to the genus actinomyces.

COMMENT

The suggestion that the condition was maceration of the skin of the feet due to hyperhidrosis was considered and rejected, as I have not seen similar rounded erosions on the feet of any of the many patients I have examined for this condition during the past two years at a tuberculosis sanatorium, where hyperhidrosis is prevalent. Furthermore, patients with hyperhidrosis manifesting a similar degree of tissue loss

Fig. 5.—High power of an area at the base of the lesion, demonstrating spores at the upper level and mycelium at the lower level.

from maceration invariably have a subacute inflammatory reaction of the soles which is distinctly noticeable, often complained of and greatly aggravated by the application of a fungicide containing salicylic acid. In the present instances inflammation was not present at any time during the periods of observation, and the conditions were benefited by the use of compound ointment of benzoic acid N. F. (Whitfield's ointment).

With regard to keratolysis exfoliativa, the only similarity that obtains is the superficial nature of both conditions. The configuration of the latter entity, which is in general considered a dermatophytid, is most irregular, as is its distribution.

SUMMARY

Two cases are reported in which there were lesions of the feet of a type not previously described. In 1 case sections showed that the tissue of the affected area was invaded by a fungus resembling Actinomyces. In the other case scrapings from the foot examined microscopically demonstrated mycelium of fine caliber.

The superficial nature of the condition would tend to place this species of Actinomyces in a category with Microsporon minutissimum and Microsporon furfur.

1930 Wilshire Boulevard.

WHEALING CAPACITY OF SKIN OF NEWBORN OR YOUNG INFANT

REPORT OF EXPERIMENTS

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AND
RUDOLF L. BAER, M.D.
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There is as yet no agreement on the question as to whether or not the skin of the newborn or young infant has the capacity to form wheals when subjected to stimuli capable of producing wheals in older persons. The decisive answer to this question seems to be of fundamental importance.

For example, the question as to whether the skin of the newborn infant is capable of whealing is of importance in relation to the nature of the wheal reaction produced in many adult skins by certain extracts commonly employed in intracutaneous or scratch testing. Such extracts, in particular those of house dust, cotton linters and human dander, are recognized as producing wheal reactions in the majority of atopic adults and even in many normal adults. But according to most authors, the same extracts, in the usual concentrations, fail to produce wheal reactions in infants under several weeks of age.

Two theories are advanced to explain this difference in cutaneous reactions. The first is that the urticarial reactions which these extracts elicit in adults are based on sensitization as a result of previous exposure; and although this cutaneous sensitization has an extremely high incidence, in the majority of instances it appears to be of no clinical significance, having no relation to specific clinical disease. According to this explanation, the newborn infant does not react to these extracts because the specific cutaneous sensitization has not yet taken place, while the common occurrence of the reaction in adults is based on the fact that this form of urticarial sensitization to particular extracts is extremely common, increasing with age and opportunity for exposure.

According to a second theory, the urticarial reaction which these extracts produce in so many adults is due to the primarily irritating

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From the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital, Columbia University.

Dr. Nathan Ratnoff and Miss Molly Gray, R.N., gave valuable assistance in this study.

or intrinsic urticariogenic property of the particular extracts; and the lack of wheal reaction after injection of the "irritating" extracts in newborn infants is due to the absence of the general capacity to wheal on the part of the newborn infants' skin. Obviously the second theory becomes untenable as soon as it is decisively shown that the skin of the newborn can produce wheals when subjected to various known primarily and intrinsically urticariogenic stimuli.

Many authors have recognized the fundamental importance of the question as to whether or not the skin of the newborn infant has the capacity to form wheals. Low [1] tested the skin of 3 normal infants (1, 10 and 13 days of age) by applying the leaf of the wild common nettle, a known urticariogenic plant; he reported that he was able to elicit whealing within two minutes after application of the leaf.

Matzinger [2] attempted to discover whether or not the newborn infant's skin is capable of whealing by passively sensitizing the cutaneous tissues of 3 babies (6 days to 2 months old) with a serum containing Prausnitz-Küstner antibodies from a patient sensitized with ascaris. In all instances perfect wheals were produced in the passively sensitized sites.

Carey and Gay [3] attempted to determine the whealing capacity of the infantile skin by intracutaneous testing with a known urticariogenic substance, namely histamine phosphate, and by intracutaneous tests with inhalant atopens after passively sensitizing the infant's skin with a serum containing Prausnitz-Küstner antibodies for the particular atopens. The results demonstrated that the infant's skin possesses a whealing capacity which, although less intense than in adults, is nevertheless definite. In the opinion of these authors, the urticarial reactions produced in infants after passive sensitization are more "variable" than in adults.

Adelsberger [4] observed that the young infant's skin is much less sensitive to intracutaneous tests with horse serum than is the adult skin. However, the author referred to the twenty-four to forty-eight hour papular type of reaction to horse serum and not to the immediate wheal type. Both Carey and Gay and Walzer [5] were apparently in error in attributing to Adelsberger the statement that she found infants less

1. Low, R. C.: Anaphylaxis and Sensitization, New York, William Wood & Company, 1925.

2. Matzinger, W.: Ueber die Askaridenallergie bei Säuglingen und die Spezifität der Askaridenreaktion, Ztschr. f. Immunitätsforsch. u. exper. Therap. **60**:399, 1929.

3. Carey, T. N., and Gay, L. N.: Skin Reactions in Infants, J. Allergy **5**:488, 1934.

4. Adelsberger, L.: Das Verhalten der Haut gegenüber verschiedenen Reizen, Ztschr. f. Kinderh. **43**:373, 1927.

5. Walzer, M.: A Critical Review of the Recent Literature on the Dust Atopen and on Vitamin C in Relation to Hypersensitiveness, J. Allergy **10**:72, 1938.

capable of urticarial reactions than were adults. On the contrary, Adelsberger stated that the "immediate wheal reaction is generally stronger in infants than it usually is in adults."

Various authors and most recently Walzer [5] have expressed the opinion that the infant's skin is significantly less capable of reacting with wheals than is the skin of the adult.

In view of these divergences of opinion and of the fundamental nature of the problem, it seemed to us proper to subject the question to further experimental study.

EXPERIMENTS

The cutaneous testing was performed on 8 full term, well developed infants, 1 of whom was 5 hours old, 4 of whom were 1 day old and 3 of whom were 2 days old. There was no family history of atopy in

Results of Intradermal Tests *

Histamine Phosphate, 1:1,000 Solution				Codeine Sulfate, 1 per Cent Aqueous Solution			
Infant		Adult		Infant		Adult	
Wheal	Flare	Wheal	Flare	Wheal	Flare	Wheal	Flare
1.1	3.9	1.1	3.7	0.7	3.4	1.1	4.5
0.7	2.9	1.3	4.7	1.0	4.0	1.7	5.0
0.9	3.5	0.6	3.3	0.7	3.5	0.7	3.3
1.0	4.2	0.7	4.1	0.7	3.8	0.6	2.9
1.6	3.6	1.7	4.1	1.9	4.4	1.7	5.3
0.6	3.7	0.9	3.5	0.7	4.5	0.9	3.0
0.9	3.2	1.5	5.0	0.7	3.8	1.8	5.1
0.6	1.2	1.0	4.0	1.2	4.3	0.9	3.9
Average							
0.92	3.3	1.1	4.3	0.95	3.96	1.2	4.1

* The largest diameter of the reaction is expressed in centimeters.

any of the babies tested. By means of a tuberculin syringe and a hypodermic needle (26 gage, ¼ inch long) the solutions employed were injected into the skin of the upper part of the back as superficially as possible. The amount injected was the smallest quantity which sufficed to produce a minute elevated area around the site at which the needle was inserted, about 0.01 cc.

The solutions injected were (1) a 1:1,000 solution of histamine phosphate (0.001 Gm. per cubic centimeter, i. e., 0.00001 Gm. of the salt in 0.01 cc.), and (2) a 1 per cent aqueous solution of codeine sulfate. The reactions were read after fifteen minutes. As controls, 8 adult patients suffering from a variety of nonatopic dermatoses were given similar injections, but in the skin of the forearm instead of the back.

RESULTS

As shown by table 1, in every infant tested and in every test performed with the two urticariogenic agents we were able to elicit definite

and typical wheal and flare reactions. Although these were on the whole somewhat smaller than those elicited in the "normal" adults used as controls, the reactions were all typical, many of the growing wheals presenting definite pseudopods. The reactions leave no doubt that the infantile skin is capable of regularly responding with an urticarial reaction on proper exposure to an urticariogenic agent. Had these reactions in the infants been elicited by common allergens, all physicians reading the tests would have considered the responses to be strongly positive.

Furthermore, it will be seen in the table that even in infancy certain persons have a greater capacity for whealing than others. Despite these variations in reactivity, which can also be observed in the adult control group, the fact remains that the skin of the newborn infant is regularly capable of producing a wheal which is comparable to that of the adult skin.

COMMENT

Our results confirm those of Low, Matzinger, Adelsberger and Carey and Gay in respect to the capacity of the infant's skin to produce wheals. In view of these results it must be concluded that the lack of urticarial reaction to cutaneous tests with ordinary allergens generally evidenced by the skin of the newborn—and even by the atopic newborn—indicates that intrauterine sensitization of the skin is not the rule. Ratner, Jackson and Gruehl [6] have based their theory of active and passive intrauterine sensitization on experiments with guinea pigs as well as on experiences and studies on human beings.

Walzer [5] has pointed out that there are several important facts which strongly oppose the theories of Ratner, Jackson and Gruehl. Walzer's criticism has lately been substantiated by the investigations of Zohn,[7] who succeeded in sensitizing 11 of 12 women in the third trimester of their pregnancy with an extract of Ascaris lumbricoides. Zohn not only elicited cutaneous reactions to ascaris extract in these patients but succeeded in producing Prausnitz-Küstner antibodies in 9 of 12 patients, while in 2 he observed a positive urticarial reaction to direct testing but found no Prausnitz-Küstner antibodies in the patient's blood serum. Blood which was taken both from the fetal cord and from the newborn

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6. (*a*) Ratner, B.; Jackson, H. C., and Gruehl, H. L.: Transmission of Protein Hypersensitiveness from Mother to Offspring: IV. Passive Sensitization in Utero, J. Immunol. **14**:291, 1927; (*b*) V. Active Sensitization in Utero, ibid. **14**: 303, 1927. (*c*) Ratner, B.: A Possible Causal Factor of Food Allergy in Certain Infants, Am. J. Dis. Child. **36**:277 (Aug.) 1928.

7. Zohn, B.: Placental Transmission of Hypersensitiveness to Ascaris Lumbricoides Actively Induced in the Pregnant Woman, Am. J. Dis. Child. **57**:1067 (May) 1939.

baby immediately after delivery did not in a single instance contain Prausnitz-Küstner antibodies. Zohn's findings confirm the work of Bell and Eriksson,[8] who were unable to elicit cutaneous reactions in the newborn babies of mothers suffering from asthma and hay fever; and even when the mothers had Prausnitz-Küstner antibodies in their blood serums, no such antibodies could be detected in the blood from the fetal cord or in the infant's blood. The findings of Smyth and Bain[9] also indicate that urticarial reactions to cutaneous tests are extremely rare in young infants, even when these infants are born of atopic mothers who have positive reactions to cutaneous tests and who have circulating reagins.

Since the bulk of previous evidence and our own findings show that the skin of the newborn infant is capable of whealing, the conclusion seems justified that the newborn infant's common lack of wheal response to ordinary allergens is based on absence of cutaneous sensitization to these allergens. This obviously speaks against the common occurrence of cutaneous sensitization in utero.

The second question to be considered in the light of these findings is the problem of whether or not the urticarial reactions produced in the many normal and atopic adults by such extracts as those of house dust, cotton linters and human dander are based on the primarily irritating and primarily urticariogenic properties of these extracts. Walzer[5] in 1938 reviewed the pertinent literature on this subject. He came to the conclusion that the common 1 or 2 plus reactions to house dust extracts seen in adults cannot be considered significant, because the majority of normal adults give such reactions; these reactions, however, cannot usually be passively transferred. Walzer mentioned the fact that infants below the age of 1 year do not usually give any reaction at all to intracutaneous tests with house dust extracts. He explained this lack of reaction as due to the "well known" diminished reactivity of the skin of children of that age to allergens as well as to histamine.

On the other hand, Walzer confirmed Hill's[10] observations that urticarial dust and feather reactions, which are passively transferable, frequently occur in children under 1 year of age who are suffering from "eczema" (atopic dermatitis). It appears to us to be difficult to reconcile the existence of such urticarial reactions in eczematous infants with the theory that infants' skins are inherently incapable of reacting with wheals

8. Bell, S. D., and Eriksson, Z.: Studies in the Transmission of Sensitization from Mother to Child in Human Beings, J. Immunol. **20**:447, 1931.

9. Smyth, F. S., and Bain, K.: The Direct Skin Test in Allergy, J. Allergy **2**:316, 1931.

10. Hill, L. W.: Sensitivity to House Dust and Goose Feathers in Infantile Eczema: The Role of Specific Allergens, J. Allergy **9**:37, 1937.

comparable to those seen in the adult. The contradiction presented by this theory is made more striking by our present findings that even the youngest and most normal infants possess the capacity to respond with wheals when their skin is exposed to adequate stimuli in the form of primarily urticariogenic agents.

On the basis of present evidence one is constrained to conclude that were the house dust and similar extracts primarily irritating or primarily urticariogenic, they should regularly produce wheals when employed in cutaneous tests, even in infants below 1 year of age, regardless of whether the infants are normal or atopic or have "eczema" or healthy skins. Since this is not the case, it becomes evident that these extracts in the proper concentrations, i. e., those ordinarily employed in cutaneous testing, are not primarily urticariogenic agents; and the wheals which they produce in adult skins with such regularity cannot be due to primary irritant or urticariogenic action but must be attributable to some other mechanism.

In analyzing the available facts it is impossible to escape the impression that some specific factor must be the basis of the wheals produced in adults by the extracts in question. For how else could one explain that certain specific allergenic but not primarily urticariogenic extracts so commonly produce whealing in adults while others do not?

Furthermore, the capacity to react with whealing to these extracts must be acquired. For how else could one explain the fact that young infants with their proved capacity for whealing still do not react to these extracts, while adults regularly respond with urticarial reactions?

Two possible explantions now present themselves, either of which could account for the specific acquired capacity of the adult skin to react with wheals to cutaneous tests with extracts which produce no wheals in the newborn. The first is that maturation itself—regardless of and without prior exposure—suffices to bring about a specific change which is at the basis of the adult capacity to react with wheals to house dust and similar extracts; the second explanation is that the specific change is based on a specific sensitization of the adult skin due to repeated exposure. For it has been shown by numerous investigators that prolonged and repeated exposures to various allergens, particularly to certain inhalant allergens, tends to produce an urticarial sensitivity to cutaneous tests in a large number of the exposed persons (Lippard, Schloss and Johnson;[11] Jadassohn;[12] Brunner;[13] Colmes, Guild and

11. Lippard, L. W.; Schloss, O. M., and Johnson, P. A.: Immune Reactions Induced in Infants by Intestinal Absorption of Incompletely Digested Cow's Milk Protein, Am. J. Dis. Child. **51**:562 (March) 1936.

12. Jadassohn, W.: Allergiestudien bei der Askaridenidiosynkrasie, Arch. f. Dermat. u. Syph. **156**:690, 1928.

Rackemann,[14] Hill,[10] Sulzberger,[15] Harkavy and Romanoff [16] and Sulzberger [17]).

It appears from observations of this type that certain allergens with a high "sensitizing index" produce specific allergic sensitivity of the urticarial type not only in atopic but in nonatopic persons, although more readily in the former than in the latter. This sensitivity may be either with or without the presence of Prausnitz-Küstner antibodies; and the presence of this form of sensitivity to cutaneous tests is often without direct clinical significance.

To recapitulate: Since the bulk of previous evidence and our own findings show that the skin of the newborn infant is capable of whealing, the conclusion seems justified that the lack of wheal response of the infantile skin to certain extracts commonly producing wheals in adults demonstrates that these extracts do not possess primarily irritating, i. e., primary urticariogenic, properties.

The common reactions in adults must therefore be based on some mechanism other than primary irritation; and the theory that these reactions are due to specific acquired cutaneous sensitivity from previous exposure to allergens with a high sensitizing index seems that which is most tenable at present.

SUMMARY AND CONCLUSIONS

Intracutaneous tests with solutions of histamine phosphate and codeine sulfate in infants 5 hours to 2 days of age reveal that even at these early ages urticarial reactions are obtained in 100 per cent of the tests. These reactions are slightly smaller than those found in an adult control group. The fact that urticarial reactions can be elicited in newborn infants with such great regularity demonstrates that all those theories are untenable which are based on the assumption that the infant's skin is not capable of a wheal response.

Since the skin of the infant under 1 month old does not usually react to common allergens, these results speak against the common

13. Brunner, M.: Immunological Studies in Human Parasitic Infestation: I. Intradermal Testing with Parasitic Extracts as an Aid in the Diagnosis of Parasitic Infestation, J. Immunol. **15**:83, 1928.

14. Colmes, A.; Guild, T., and Rackemann, F. M.: Studies in Sensitization: Influence of Occupation on Sensitization in Man as Determined in a Study of Thirty-Two Bakers, J. Allergy **6**:539, 1935.

15. Sulzberger, M. B.: Recent Immunologic Studies in Hypersensitivity to Tobacco, J. A. M. A. **102**:11 (Jan. 6) 1934.

16. Harkavy, J., and Romanoff, A.: Skin Reactions to Tobacco and Other Allergens in Normal Men and Women Smokers, J. Allergy **6**:56, 1934.

17. Sulzberger, M. B.: Allergy in Dermatology, J. Allergy **7**:385, 1936.

occurrence of intrauterine cutaneous sensitization. Moreover, our results oppose the theory that the common urticarial cutaneous reactions of adults to tests with certain extracts, such as house dust, cotton linters and human dander, are due to the primary irritating properties of these extracts and indicate that the common occurrence of wheal reactions to these extracts in adults is based on the high incidence of a certain form of cutaneous sensitization to these particular agents.

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ERYSIPELOID

REPORT OF A CASE

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Erysipeloid is an acute infectious disease caused by an actinomyces organism, Erysipelothrix rhusiopathiae. It is usually localized in the skin but may present the picture of a severe systemic disease with manifold constitutional symptoms. The organism is saprophytic on fish slime and putrefying material and produces a disease in hogs called swine erysipelas. It is our purpose in presenting this paper to call attention to a disease which is probably more frequent than is generally recognized and to report an example which we believe is somewhat unusual but nevertheless characteristic of one of the clinical types of erysipeloid.

The original description of the disease is ascribed to Fox,[1] who in 1873 reported on a cutaneous disease in a man who tried on boots made of sheep skin. The infection began on a finger and spread to the remainder of the hand, which became swollen and red but did not suppurate. He considered the dye used on the leather to be the cause. Later in the same year a better description was given by Baker,[2] who called the infection erythema serpens. In 1884, and later in 1887, Rosenbach [3] gave a complete description of the localized condition and applied the name erysipeloid. He isolated an organism which was almost certainly the cause of the disease and reproduced the infection on his own arm by inoculating himself with cultures of material from a patient who contracted the condition from foul cheese.

In this country Klauder and his associates [4] have made most complete and exhausting studies of erysipeloid. The original descriptions of the

1. Fox, W. T.: Skin Diseases: Their Description, Pathology, Diagnosis, and Treatment, ed. 3, London, H. Renshaw, 1873, p. 108.

2. Baker, cited by Klauder.[4b]

3. Rosenbach, F. J.: Ueber das Erysipeloid, Verhandl. d. deutsch. Gesellsch. f. Chir. **16**:75, 1887.

4. (*a*) Klauder, J. V.; Righter, L. L., and Harkins, M. J.: A Distinctive and Severe Form of Erysipeloid Among Fish Handlers: Report of Clinical and Laboratory Studies, Arch. Dermat. & Syph. **14**:662 (Dec.) 1926. (*b*) Klauder, J. V.: Erysipeloid and Swine Erysipelas in Man: A Clinical and Bacteriologic Review; Swine Erysipelas in the United States, J. A. M. A. **86**:536 (Feb. 20)

(Footnote continued on next page)

disease were of the localized form, and reports of erysipeloid of the more severe variety did not appear until later. The description that follows is taken largely from the various publications by Klauder.

Erysipeloid is most frequently an occupational disease. It occurs in those who handle fish and crabs, in veterinarians, butchers, cooks and bone button workers. It may occur in any one who injures himself while working with fish or with putrid or decaying matter.

Swine erysipelas is a common disease in Germany, and the literature of that country abounds in descriptions of erysipeloid in human beings and of the infection in hogs. In hogs it is always a systemic infection presenting three types: the mild, the severe and the chronic. The same types have also been described as appearing in human beings. In swine the disease may present, in the mild form, bluish red cutaneous lesions, with mild constitutional symptoms; in the severe form, cutaneous lesions associated with polyarthritis and septicemia, and in the chronic form, polyarthritis. Swine erysipelas is on the increase in the United States.

In this country Gilchrist,[5] at a meeting of the American Medical Association in 1896, first described the disease in crab handlers. He gave a report of 156 cases of the localized type of the infection examined at Johns Hopkins Hospital. He did not isolate an organism. The character of the disease was apparently not recognized by him until it was pointed out in the discussion of his paper as erysipeloid by Dr. C. W. Allen of New York. Klauder made his first report in 1926.[4a] In 1930 Ritchie and Becker[6] reported a case from the Great Lakes region and called attention to the connection of the disease with fresh water fish. Klauder's patients came chiefly from the Chesapeake Bay area and the New Jersey coast. In 1933 Lawson and Stinnett[7] reported 247 cases of erysipeloid among persons working in a bone button factory. In 237 of them the condition was a typical localized cutaneous infection; in 7 infections of the eyes occurred, and in 3 workers who inhaled bone dust the infections were bronchial. The bones used were cattle bones from Europe and South America, imported through Chicago. In September 1934 Light[8] reported a case in which the condition occurred in a meat handler. The infection appeared twelve days after injury. The

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1926. (*c*) Klauder, J. V., and Harkins, M. J.: Erysipeloid in the United States: Clinical and Laboratory Studies, J. A. M. A. **96**:1205 (April 11) 1931. (*d*) Klauder, J. V.: Erysipeloid as an Occupational Disease, ibid. **111**:1345 (Oct. 8) 1938.

5. Gilchrist, T. C.: M. Rec. **11**:783, 1896.

6. Ritchie, E. B., and Becker, S. W.: Erysipeloid: Report of Case of Infection from Fish of Great Lakes, J. A. M. A. **95**:26 (July 5) 1930.

7. Lawson, G. B., and Stinnett, M. S.: Erysipeloid Occurring Among Workers in a Bone Button Factory, South. M. J. **26**:1068, 1933.

8. Light, S. E.: Erysipeloid: Report of a Case, Northwest Med. **33**:331, 1934.

hand only was involved. Subsequently the epitrochlear and axillary glands became swollen, but there was no fever.

The localized form is the most frequent type of the disease. The incubation period is one to four days. The first evidence of the infection is local redness, pain and swelling at the site of an injury, such as that caused by the prod of a fish fin. The swelling and erythema quickly spread by contiguity and through the lymphatics and produce a purplish red area, with clearly defined, slightly raised borders. As the lesion progresses the center fades, producing a characteristic appearance; it varies in size and shape, but the borders are sharply demarcated and slightly raised, and the color is purplish red at the borders, fading to salmon pink at the center; it is tender and usually warm to the touch. If the infection begins on a finger, it may spread to the hand and in turn may involve each finger and the dorsum of the hand and even extend up the arm. The parts may be immobilized by the swelling. The pain is usually described as a burning, itching sensation, which is usually rather mild but may be intense.

Lymphangitis, with swelling of the regional glands, occasionally occurs in the more severe variety of the localized form. Suppuration never occurs, nor does desquamation after the infection subsides. The disease may last for five days to two months or longer and will usually subside spontaneously, even if not treated. The migration of the infection is characteristic. The original lesion may fade, and the condition recur in another place or in the same locality and again pursue the original course.

The chronic form of the infection may last three to eight months or longer. The severe variety in human beings follows closely the pattern of the disease in swine. It starts as the result of an injury, with the usual localized cutaneous manifestations, but is associated with fever, generalized malaise, headache and migratory symptoms in the joints. The cutaneous lesions rapidly spread, fade, disappear and recur in other and varied parts of the body. The case to be reported is characteristic of this phase of the infection, and more detailed description will be included in the report. Septicemia and death, with an endocarditis-like infection, have been described.

The organism is a pleomorphic gram-positive Coccobacillus which belongs to the family Actinomycetaceae. It is called Erysipelothrix Rosenbach, Erysipelothrix rhusiopathiae or Erysipelothrix rhusiopathiae suis. It was described by Ford [9] as an anaerobe which grows best in gelatin, occurring first as a small gram-positive coccus or rod that later develops into long, threadlike forms. True branching has been described. Stitt did not mention its anaerobic qualities. Nowhere did

9. Ford, W. W.: Textbook of Bacteriology, Philadelphia, W. B. Saunders Company, 1927.

Klauder state that it is an anaerobe, nor did Rosenbach in his original article describe it as such. It is found in the dejecta of hogs and is the cause of swine erysipelas. In 1889 Hillebrand and Mayer [10] first reported the occurrence of swine erysipelas in human beings. The organism is found in the colons of hogs, and virulent organisms have been recovered from the gallbladders of swine by Pitt.[11] The organism recovered from fish slime and occurring in almost any putrefying protein or nitrogenous material and known to cause erysipeloid has been found by numerous authors to be similar to the organism causing swine erysipelas. Klauder stated the belief that the organism is the same, as well as that which causes erysipelas in mice, Bacillus murisepticus. Evidently, the various changes through which the organism is obliged to pass to survive in its natural state are responsible for the varied descriptions of its characteristics. It is possible that the organism is a facultative anaerobe that can be caused gradually to grow in the presence of oxygen under certain circumstances. It is highly virulent for pigeons and mice but does not produce infection in sheep and guinea pigs or in other domestic and laboratory animals, such as cats and dogs.

In treatment of the local lesions the time-honored stand-bys of rest and moist heat are the most effective. Many and varied preparations have been recommended; ichthammol and iodine were used by the early writers. Klauder [4d] suggested the constant use of dressings wet with 12 per cent ichthammol in alcohol and repeated erythema doses of radiation with a water-cooled quartz mercury vapor arc lamp. He also stated the belief that local treatment is of doubtful value and that the disease runs a self-limited course in spite of the treatment. Immune serum may be effective for the more severe conditions. Klauder recommended its use in cases in which articular symptoms are present, in those in which the disease lasts longer than one month and in cases in which the spread is rapid and there are severe constitutional symptoms. The serum may be injected intramuscularly, intravenously or in the site of the lesion. The total dose may be given over a period of from twenty-four to seventy-two hours.

REPORT OF CASE

J. B. T., a white man aged 49, was admitted to Norfolk General Hospital as a patient of Dr. P. St. L. Moncure Sept. 9, 1938, after an accident in which his automobile was struck by a streetcar. He had suffered a blow on the head and a slight laceration of the right knee. He was treated for what were considered minor injuries and was discharged from the hospital on September 12. He was readmitted four days later, with the history that he had felt well until the day before, when his right knee had become painful and a small amount of pus had been expressed from the wound. A high fever, coryza and generalized aching of the bones and

10. Hillebrand and Mayer, cited by Klauder.[4b]

11. Pitt, cited by Ford.[9]

muscles then developed. There appeared at this time a red painful area about the lesion on the knee and a generalized rash on his body and limbs which was painless and did not itch.

His past history was of some importance. A stone had been removed from the left kidney fifteen or sixteen years before. He had typhoid fever when a child. He gave a previous history of headaches and sinusitis but none in the past five to six years. He had influenza in 1918 and had had a chronic cough since that attack. He had suffered with attacks of pain in the upper part of the abdomen for the past five or six years. The attacks occurred every four to six weeks, were severe and cramplike and radiated to the back, and he frequently required hypodermic injections of an anesthetic for relief. There was nausea but no vomiting. There was no history of jaundice. He had had gonorrhea twenty-five years before but reported no symptoms of syphilis. He had no symptoms referable to the bladder. He had complained of mild rheumatic symptoms since childhood.

On examination at the time of his second admission his skin was hot and dry. Over his trunk and extremities there were many ovoid or round erythematous spots, varying in size from that of a quarter to that of a silver dollar, with clearly defined edges. They were not tender. There was a healing abrasion of the right knee, surrounded by a tender hot red area about the size of one's hand, the edges of which were well demarcated. The patella rode rather high, as if fluid were beneath. On deep pressure there was much tenderness on the right side of the patella. Below the patella there was a swollen, puffy area. There was a slight abrasion of the scalp. The eyes and the nose presented no gross pathologic changes. The antrums were dull on transillumination. The lower teeth were out. The few remaining upper teeth did not appear diseased. The throat was slightly red and injected. The muscles of the back of the neck were stiff and sore. The heart presented no unusual changes. The blood pressure was 120 systolic and 70 diastolic. There were some moist rales and impaired resonance at the base of the right lung. The liver was palpable 2 fingerbreadths below the right costal margin. The spleen was not felt. There was a scar over the region of the left kidney. Rectal examination revealed no pathologic changes. The reflexes were not pathologic. His temperature was 102.4 F.; the pulse rate, 90 and regular, and the respiratory rate, 20.

Laboratory examination showed 90 per cent hemoglobin, and 4,600,000 erythrocytes and 10,000 leukocytes per cubic millimeter, with 91 per cent polymorphonuclear neutrophils, 7 per cent lymphocytes and 2 per cent monocytes. The Wassermann reaction was negative. The Widal reaction was negative. The blood culture gave negative results. The sputum culture showed chiefly staphylococci and a few colonies of Streptococcus viridans. Examination of the urine showed a specific gravity of 1.021, a trace of albumin, no sugar and an occasional white blood cell.

He was thought to have erysipelas or an erysipelas-like infection with toxic erythema and was given sulfanilamide, 90 grains (5.82 Gm.) a day for one day and then 60 grains (3.88 Gm.) a day for two days. Local treatment consisted of hot wet dressings of saturated boric acid solution. This therapy was followed by a complete subsidence of the fever, the clearing of the signs in the chest and a disappearance of the rash. The sulfanilamide was discontinued after the third day. Three days later the patient complained of a sore, tender spot at the base of his spine. On examination, he was found to have on the coccyx a reddish tender spot, warm to the touch, with clearly defined edges. The following day his temperature rose to 101.6 F., and he was again given sulfanilamide. The lesion promptly cleared. Tenderness subsided, and in four more days, on September 28, he was discharged but continued to take small doses of sulfanilamide.

The patient was readmitted three days later, with the history that he had felt well for about two days but that the day before, a spreading red sore area had appeared on his left foot and extended up to below the knee.

The examination on this admission gave results similar to those of the previous visit except that the skin over the left foot and leg was red and tender and hot to the touch. The edges of the lesion were slightly raised and sharply defined. The skin gave the appearance of being slightly swollen, but there was no definite edema. There were a few rales at the bases of the lungs.

On this admission no significant changes had taken place in the laboratory observations. Examination of the blood showed 83 per cent hemoglobin, and 3,760,000 erythrocytes and 10,700 leukocytes per cubic millimeter.

The patient was again given sulfanilamide because of the previous experience, and hot wet boric acid dressings were applied locally. The temperature promptly returned to normal, but five to six days elapsed before the area on the foot and leg subsided. He then began to complain of painful elbows. Areas appeared over the elbows like those already described. On October 8 an area developed on the right knee; on October 14 the area on the right knee disappeared, but he complained of soreness of the left arm. Spots then occurred in various places over the body. They took on a more dull purplish red, with central fading. He complained from time to time that his joints were so painful that he could not move the affected limbs. All the symptoms persisted in spite of sulfanilamide, and he began to have a temperature of 100 to 101 F. while the drug was being given. Administration of the drug was therefore discontinued on October 19. During this time his spleen had become palpable, and the liver had enlarged. Anemia developed, necessitating the administration of iron and liver and blood transfusions. He also complained of severe pain in the region of his gallbladder, and on November 2 he suffered a severe attack of pain in this region.

The clinical picture at this time was puzzling, and the exact nature of the infection seemed to us uncertain. We thought that he had some type of erysipelas associated with a focus of streptococcic infection deeply situated in a vein or in the liver. He was therefore given sulfanilamide by subcutaneous injection for three days, but without apparent improvement (500 cc. of 0.8 per cent solution was given the first day; then 300 cc. of 0.8 per cent solution twice a day for two days). All treatment, except local, symptomatic and supportive, was then discontinued. The patient continued the same course. His temperature ranged between 100 and 101 F., sometimes going to 102. We then noticed the similarity of the clinical picture to that in certain cases described by Klauder, and on November 27 and 28 we gave the patient 200 cc. of erysipeloid antiserum.[12] After this there was definite improvement; the fever and the general symptoms subsided. On December 6 a rise in temperature again occurred, and he complained of pain in the right side of the chest. Examination revealed signs of pleurisy at the base of the right lung, which was confirmed by a roentgenogram. This infection also subsided; in about five days he became afebrile and asymptomatic, and on December 18 he was discharged.

Four days after discharge soreness, redness and swelling of his right foot developed, with intermittent swelling and tenderness of the toes. He then began to have all of his former symptoms again but less severely and without fever. On Jan. 3, 1939, a roentgenogram of his gallbladder showed it to be filled with stones. Since this was thought to be a possible focus of infection, he was readmitted to the hospital, and on January 31 Dr. Moncure removed a widely distended gall-

12. Erysipeloid antiserum was furnished by Sharp and Dohme.

bladder filled with stones. The patient made an uneventful recovery from the operation but occasionally continued to have some erythematous areas on his skin. These gradually disappeared, and when last seen (in June 1939) he had been free from all symptoms for several months.

The culture of the bile removed from the gallbladder gave no growth. Anaerobic culture of the wall of the gallbladder resulted in a growth of small gram-positive pleomorphic coccobacilli. After six months the cultures have begun to show filament formation.

The patient's blood serum agglutinated this organism completely in a dilution of 1 to 320. This rather high degree of immunity to the organism isolated argues strongly in favor of its being the specific cause of the infection in this person. Later attempts to agglutinate the organism with a specific antiserum obtained from Sharp and Dohme were unsatisfactory, because the organism clumped spontaneously at all dilutions, both in the antiserum and in the blood serum from the patient. This reaction we thought to be due to the tendency to filament formation.

SUMMARY

This patient presented a rather typical example of generalized infection due to the organism known as Erysipelothrix rhusiopathiae. The specific nature of the infection is indicated by the growth characteristics, staining quality and appearance of the organism. It is further supported by the specific agglutination of the organism by the patient's own serum in high dilution. While repeated blood cultures gave negative results, it is evident that the infection passed through the blood stream to distant parts of the body from the portal of infection in the skin over the right knee. Fresh areas of cutaneous infection developed in distant parts of the body not directly connected by lymphatic drainage with the original focus. The organism was eventually isolated from the gallbladder. The clinical course of the disease simulated closely erysipeloid in swine. This is especially interesting since the organism is also found in the gallbladders of swine. Especially striking were the manifestations in the joints.

Treatment with sulfanilamide was apparently ineffective. The use of specific antiserum was followed by definite improvement, both in the general symptoms and in the local lesions of the skin and joints. It was not until the focus in the gallbladder was removed, however, that the patient made a complete recovery.

In this case the actual source of the infection is not known, but the widespread distribution of the organism in nature makes it possible for the infection to be contracted almost any place.

We wish to call attention to the fact that the disease commonly known as "fish poisoning" is a localized form of erysipeloid. It is usually localized in the skin but may be severe, producing profuse morbidity and economic loss. The occupational characteristics of the disease must not be lost sight of.

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ERUPTIONS AND PHOTOSENSITIVITY DUE TO DYED FABRICS

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Dyed furs and leather (shoes and hatbands) have long been recognized as causes of cutaneous toxicity. Dermatitis venenata due to idiosyncrasy to dyes in other apparel has received less attention, although this phase of the subject has been considered in articles to be mentioned later. Personal studies indicate that additional lesions may be produced by such dyes. In fact, these agents may lead to the development of photosensitivity, furunculosis and possibly hidradenitis suppurativa, lichen simplex chronicus (Vidal) and other entities.

DERMATITIS VENENATA

Simple contact dermatitis constitutes the most common manifestation of sensitivity to dyes. In 5 per cent of the cases of dermatitis venenata in my private practice the condition is caused by such agents. Cole,[1a] as the result of a questionnaire, found that of 514 cases of dermatitis due to various dyes, cosmetics and furs, in 56, or nearly 11 per cent, the condition was caused by dyed clothing.

Dress Dermatitis.—Dress dermatitis is not uncommon. Goodman and Sulzberger [1] discussed 25 proved cases and stated that they had observed an equal number of probable but unproved instances. Tobias [2] studied 18 similar examples, while Bonnevie and Genner [3] listed 13 more.

In the cases reported by Goodman and Sulzberger,[1] there were 30 positive reactions to patch tests with black materials, 9 with brown, 8 with red, 5 with green and 2 with orange. Sixteen of the 25 patients tested reacted to "pure" commercial dyes on cutaneous testing. The

1a. Cole, H. N.: Investigation of Injuries from Hair Dyes, Dyed Furs and Cosmetics, J. A. M. A. **88**:397 (Feb. 5) 1927.

1. Goodman, J., and Sulzberger, M.: Acquired Specific Hypersensitivity to Simple Chemicals: I. Eczematous Sensitivity to Clothing and to Cosmetics with Special Reference to Dyes, J. Allergy **9**:136 (Jan.) 1938.

2. Tobias, N.: Dermatitis from Wearing Apparel, Internat. Clin. **3**:130 (Sept.) 1938.

3. Bonnevie, P., and Genner, V.: Eczema Due to Dyed Clothing, Arch. Dermat. & Syph. **34**:220 (Aug.) 1936.

authors frequently encountered polysensitivity. This they illustrated in a patient who reacted to seven different shades or colors. Tobias [2] found that in 15 of his cases the condition was due to black dresses, while in 2 it was due to red and in 1 to tan. In all of Bonnevie and Genner's [3] cases the condition was caused by blue dyes.

Burgess [4] listed 6 examples of dress dermatitis, all due to new silk crepe dresses. All his patients manifested positive reactions to patch tests performed with the suspected material. Thomas [5] observed 2 cases in which the condition was due to brown and orange-brown dresses. The eruption in Valade's [6] patient resulted from a black crepe dress. Garnier, Chapireau and Bourdeau [7] studied a patient in whom erythema developed after the wearing of a dress dyed with aniline black, apparently a common offender.

The essential characteristics of this form of dermatitis are well illustrated by the following typical case.

CASE 1.—Mrs. G. S., a 53 year old obese housewife, purchased a new blue rayon dress for $1.00. After she wore it for one week a pruritic erythematopapular eruption developed under her arms and soon spread to the back of her neck and to her cubital fossae. She stated that on the day the eruption appeared she had been warm and had perspired a great deal. The reactions to patch tests with material from the dress were rated as 4 plus, and wearing of the dress was discontinued. A soothing ointment was prescribed for local use. However, a full month was required for complete disappearance of the eruption.

The dress was soaked overnight in cold water, without resulting in any discoloration of the water. The following morning it was washed in warm water and washing powder, and the water became dark blue. A patch test with material from the washed dress gave a negative result, and further wearing of the garment was not accompanied by a recurrence of the eruption.

The features illustrated by this case and by the previously reported ones can be summarized. As a rule, the patient is an obese middle-aged woman. In Tobias' [2] 18 cases the average weight was 151 pounds (68.5 Kg.). In 5 patients he found the basal metabolic rate to be —12 per cent. The incriminated fabric is usually a cheap one, suggesting that inferior dyes or dyeing processes may be a factor in these instances. The noxious clothing is worn usually for some time prior to the onset of the dermatitis. This may be due to an incubation period in the

4. Burgess, J. F.: Contact Eczema from Dyed Clothing, Canad. M. A. J. **37**: 237 (Sept.) 1937.

5. Thomas, M. S.: Dermatitis of the Axillae Due to Paraphenylenediamine, Brit. J. Dermat. **42**:449 (Oct.) 1930.

6. Valade, C. K.: Contact Dermatitis from Black Crepe Dress, Urol. & Cutan. Rev. **41**:115 (Feb.) 1937.

7. Garnier, G.; Chapireau, and Bourdeau: Eruption due à une robe teinte au noir d'aniline, Bull. Soc. franç. de dermat. et syph. **43**:92 (Jan.) 1936.

development of sensitivity or to the necessity of copious perspiration for the development of the eruption. It usually requires from two weeks to six months for the complete disappearance of the exanthem after the discarding of the garment. This chronicity may be due to the retention of the dye in the skin. The possibility of temporary polysensitivity causing prolongation of the process must also be considered.

Certain clinical characteristics of dress dermatitis should cause one to suspect the etiologic significance of the patient's clothes. The eruption commonly appears first in the axillas and then spreads to the neck, cubital fossae and wrists. The last location is involved if the dress is tight about the wrists. The exanthem may progress to involve the entire trunk and extremities, although it usually spares those portions of the cutaneous surface covered by undergarments. The dermatitis usually consists of erythema with superimposed closely set papules. Pruritus tends to be severe. The vault of the axilla is often spared, the eruption involving the periphery mainly.

Other Types of Contact Dermatitis.—Obviously dermatitis venenata due to other dyed clothing is basically identical with that described under dress dermatitis. The only difference is in the sex of the patient and in the location of the eruption. The lesions in the following patient could be classified as dress dermatitis, except that they occurred in a man and were due to blue polo shirts and to a blue sweater.

CASE 2.—P. T., a 49 year old officer in the British reserve, was seen on April 8, 1939, complaining of a pruritic eruption of two months' duration. The lesions involved the axillas and neck. Patch tests elicited strongly positive reactions to a dark blue polo shirt. The reactions were slightly less positive to a light blue polo shirt and to a blue slip-on sweater. Tests performed with a blue cotton shirt and with a blue coat sweater gave negative results. The dark blue polo shirt was boiled, and the blue water so obtained was employed as patch test material. The patient reacted strongly to even this dilute extract of the dye. A patch test performed with the gray material remaining after the complete extraction of the dye by boiling in water failed to elicit any reaction. About six weeks was required for complete subsidence of the eruption after the cessation of wearing all blue shirts and sweaters.

The following patients demonstrate the necessity of recognizing the causative factor. In case 3 injudicious therapy led to unpleasant complications. This is not surprising when one realizes that such patients are often polysensitive.

CASE 3.—A 6 year old girl was given a pair of blue canvas tennis shoes. Several days after she started wearing them a dermatitis appeared on the dorsa and sides of her feet. The mother treated the lesions with saponated solution of cresol U. S. P., but when they became more aggravated she took the child to a dermatologist, who considered the eruption to be a dermatomycotic infection.

He prescribed a strong parasiticide, the application of which was followed by generalization of the eruption. Despite ten days' hospitalization, the patient required over a month for complete healing.

Case 4.—A 21 year old bank clerk had a dermatitis of his hands and legs of two years' duration. An allergist, who had studied him before I saw him, noted that the eruption improved when he was away from work but recurred on his return to work. Naturally, industrial hazards were investigated, with negative results. A patch test with material from a brown suit which he always wore to work gave a positive result. Discarding the suit resulted in immediate improvement, although several months was required for complete clearing of the lesions.

White [8] in 1897 recorded 5 instances of dermatitis due to unwashed black cotton shirts. Aniline black was the incriminated dye in these cases. Killick and Ingram [9] observed a case of dermatitis due to a redyed black blouse. They were unable to identify the dye, but they did determine that it contained no metadiamines, paradiamines or paraaminophenols. Bonnevie and Genner [8] recorded 1 case in which the dermatitis was due to a blue coat. Both these authors and Goodman and Sulzberger [1] reported lesions due to suits. Porosz [10] noted 2 patients with dermatitis of the legs due to aniline black—dyed socks. Sézary and Deval [11] wrote on dermatitis caused by black gloves. Sézary, Lefèvre and Rivoire [12] saw a dermatitis of the neck develop after contact with a red and yellow handkerchief dyed with azarine S and azofluvine. Red pajamas were at fault in 1 case mentioned by Goodman and Sulzberger.[1] According to Schwartz,[13] dress shields may cause similar eruptions. Chipman [14] observed a number of cases in which the thighs and hips were involved, as the result of using a dye at home in tinting undergarments.

Simon and Rackemann [15] reported an extensive example of polysensitivity to dyes. Dr. Simon, incidentally, was the patient. Dermatitis

8. White, cited by Killick and Ingram.[9]

9. Killick, E. M., and Ingram, J. T.: Dermatitis Following the Wearing of Dyed Fabrics, Lancet **1**:77 (Jan. 14) 1933.

10. Porosz, M.: Eczema from Dyes in Stockings, Dermat. Wchnschr. **78**:510 (May 3) 1924.

11. Sézary, A., and Deval, L.: Dermite artificielle causée par des gants nous, Bull. Soc. franç. de dermat. et syph. **39**:1370 (Nov.) 1932.

12. Sézary, A.; Lefèvre, P., and Rivoire: Dermite artificielle du cou consécutive au contact d'un foulard teint, Bull. Soc. franç. de dermat. et syph. **37**:1316 (Dec.) 1930.

13. Schwartz, L.: Skin Hazards in American Industry: Dermatitis from Wearing and Handling Dyed Fabrics, Public Health Bulletin 229, United States Treasury Department, Public Health Service, 1936.

14. Chipman, E. D.: Dermatitis from Dyed Garments, Arch. Dermat. & Syph. **33**:537 (March) 1936.

15. Simon, F. A., and Rackemann, F. M.: Contact Eczema Due to Clothing, J. A. M. A. **102**:127 (Jan. 13) 1934.

developed from the following objects: a blue serge suit, a brown suit, a dark blue overcoat, black lisle and black rayon socks, a hatband, garters, shoes, bed clothing, automobile upholstery, varnish on a mahogany table, linings of two suits, his wife's dresses and the upholstery on his living room furniture. Patch tests gave negative results with undyed wool, cotton and silk fabrics and to four commonly used mordants. However, positive reactions developed to patch tests with fourteen of thirty-two commercial "pure" dyes. The Prausnitz-Küstner reaction was negative. Simon [16] later told of a dermatitis on his feet which developed from walking barefooted on a red-dyed staircase. He reported that he was sensitive to azo dyes.

Industrial Aspects.—At times contact dermatitis due to dyes in clothing may be of industrial significance. According to Schwartz,[13] of 1,600 cases of industrial dermatitis included in the combined statistics of New York and Ohio in 1934, in 45 (2.8 per cent) the eruption was due to dyes in fabrics. He included models, saleswomen, checkroom attendants, clothes pressers and dressmakers in the list of those affected. Goodman and Sulzberger [1] listed 2 such cases. One was in a female sewing machine operator who was sensitive to paraphenylene diamine and who reacted to seven black and two brown materials with which she worked. They also proved a male sewing machine operator to be sensitive to red crepe. In 1 of the patients studied by Bonnevie and Genner [3] an eruption developed on the hands and forearms from washing blue clothes.

In 2 cases in my practice uniforms provided the industrial hazard; one was a bellboy's uniform, and the other (case 7), a mailman's shirt.

PHOTOSENSITIVITY

The most unusual effect of dyed clothes on the skin is the development of photosensitivity. I was unable to locate any previous reports of this complication. Sandler [16a] reported an instance of pigmentation apparently due to water squeezed out of a wet red bathing suit. This condition, however, does not correspond to the type of photosensitivity seen in these 2 patients. It is recognized that certain dyes, including tetrabrom-tetrachlor-fluorescein, eosin, acriflavine hydrochloride and methylthionine chloride (methylene blue), may sensitize the skin to solar radiation. Protection from sunlight is one of the prime functions of clothes. Therefore, it is interesting to note that occasionally they may have the opposite effect.

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16. Simon, F. A., in discussion on Goodman and Sulzberger.[1]

16a. Sandler, I. L.: Photosensitizing Agents, J. A. M. A. **112**:2411 (June 10) 1939.

CASE 5.—J. B., a 25 year old music student, referred by Dr. Newton Shapiro, on July 24, 1939, started to wear a new pair of rayon pajamas with red and purple stripes. According to the manufacturer, vat dyes had been employed in coloring the material. Five days later he noticed some pruritus of his penis and scrotum and visited a general practitioner, who prescribed a coal tar ointment. This he applied only once. On the way to the physician's office he wore a pair of glasses but left his head uncovered. He noticed that the sun seemed particularly warm and that his face itched and burned a little. The following day his ears, eyelids and genitalia began to swell. Pruritus was intense. When seen on the following day the patient presented severe erythema and edema of his ears, eyelids, cheeks and nose. The portion of his nose covered by the nose piece of his glasses was free of lesions. His penis and scrotum were greatly reddened and swollen. On his arms, legs and trunk were a number of small discrete vesicles with erythematous areolas. Constitutional symptoms were not pronounced. The patient's temperature was 98.2 F. by mouth. The urine he passed was no darker than normal.

Therapy consisted of rest in bed, in soothing applications, wet dressings and starch and soda baths. He was completely well in two weeks, despite the complication of a generalized maculopapular eruption believed to be due to pentobarbital sodium.

On August 22 the patient reported a mild recurrence of the dermatitis of his genitalia after wearing the same pajamas. The results of patch tests with material from the stripes were positive and with material from the white portion, negative. There has been no further difficulty since he discarded these pajamas.

CASE 6.—E. P. L., a 64 year old dentist, was first seen on Nov. 4, 1939. About two weeks previously he had worked in the garden wearing a pair of dark blue trousers, which had cost $1.75 and which he had been wearing twice a week for three or four months, without trouble. The following day a severe dermatitis of his genitalia developed, characterized by severe edema and erythema of his penis and scrotum. His urine was not darker than normal. Dr. Oppenheimer treated him with wet dressings and soothing lotions, and he improved rapidly. On November 2 he again wore the blue trousers and worked in the garden. On each occasion, he had also worn a hat with a medium-sized brim. Before finishing his outdoor activities on the second day, he was conscious of an itching and burning sensation of his face.

When seen two days later, the dermatitis of his genitalia had relapsed. His face was involved in a papular urticarial eruption sparing his forehead and eyelids, which had been protected by his hat. The eruption cleared within three days with conservative therapy. A patch test performed with material from the suspected pants resulted in a 4 plus reaction.

In both cases the eruption cleared rapidly, differing from the usual dye dermatitis in this respect. It is interesting that the male genitalia were the primary irritated parts in each instance. One can only speculate as to whether the androgens have any relation to photosensitivity. Lancaster [17] expressed the opinion that the estrogenic hormone may be of significance in light-produced dermatoses.

17. Lancaster, A. H.: Estrogenic Hormone Therapy in Sunlight Eruptions of the Female, South. M. J. **32**:495 (May) 1939.

PYODERMA

Pyogenic cutaneous complications are not unusual in eruptions due to dyed fabrics. It is not clear just why this should occur. The most plausible explanation is that scratching initiates the infection. It is also possible that dye in the skin may act as an irritant and lead to the development of furunculosis by lowering the resistance of the skin to staphylococci and streptococci.

Goodman and Sulzberger [1] stated that in several of their cases axillary abscesses and furuncles occurred. Their opinion is well expressed by the following quotation, which they published in italics: "We believe that hypersensitivity to dress materials etc., may even play a part in the causation of certain cases of hidradenitis appearing without manifest dress dermatitis." The following case tends to confirm this viewpoint.

CASE 7.—C. L. F., a 28 year old letter carrier, referred by Dr. Sadie E. Berkove, gave a typical history of recurrent dermatitis of his neck and right axilla occurring on hot days on which he wore his bluish gray mailman's shirt. For about one month before he was seen he had noticed more or less continuous erythema and pruritus of his right axilla. He felt that he perspired more under the right arm than under the left. Furthermore, he carried the strap of his mail bag in such a manner that it pressed his shirt against his right axilla. About the end of September 1939 a few boils developed, confined to his right axilla.

He was seen on October 9, presenting multiple deep closed and draining abscesses in this location, associated with cordlike induration. Morphologically, the eruption was indistinguishable from that seen in hidradenitis suppurativa. Therapy consisted of roentgen ray irradiation, sulfanilamide, sedation, infra-red irradiation, injections of staphylococcus toxoid, surgical drainage and compresses wet with alibour water (water containing zinc and copper sulfate). He made a rapid recovery, being considered well on October 26.

Two weeks later the patient returned with a similar involvement of his left axilla. He had been carrying the bag under this arm, rubbing the dye into the skin. These lesions healed in two weeks. After this he was transferred to a truck, eliminating the necessity of carrying a bag. However, he continued to wear gray shirts. This resulted in several recurrences in each axilla. He did not completely recover until he was given employment inside the postoffice, where he was able to wear white shirts. No further contact dermatitis was noted after the onset of the furuncles.

One would suspect involvement of the apocrine glands in this case. Perhaps the chronicity of hidradenitis suppurativa may be due to the continued wearing of articles of clothing colored with a dye to which the patient is sensitized.

Valade [6] and Burgess [4] also noted pyogenic complications in dress dermatitis.

OTHER CUTANEOUS LESIONS

Goodman and Sulzberger [1] found that 1 of their patients was cured of chronic urticaria by the elimination of certain dyed clothing. This

seems logical, as an allergen often causes different manifestations in different persons.

The cause of lichen simplex chronicus (Vidal) is poorly understood. The following case suggests that occasionally dyed clothing may be of significance.

CASE 8.—L. L., a 25 year old Chinese man, was first seen on June 16, 1938, because of a pruritic erythematosquamous eruption of his face and neck. He had been using a sulfur ointment, which probably resulted in aggravation of the process. The lesions had started on his neck two months previously. It was noted that the eruption was most severe on that portion of the neck in contact with a blue sweater above his shirt collar. A patch test was performed with a sample of the sweater, and a positive reaction was obtained. Soothing applications, roentgen therapy and elimination of the sweater resulted in a "cure" after approximately one month.

However, the patient is apparently unable to understand that the eruption is caused by the sweater. As a result, he persists in wearing it, resulting in frequent recurrences of clinically typical lichen simplex chronicus, consisting of patches of flat, discrete, closely set papules with lichenification. The lesions invariably occur on the sides and back of the neck and on the elbows, where the sweater is in close contact, and usually subside in about one month.

COMMENT

It is apparent that eruptions due to dyed clothing occur frequently enough to be worthy of serious consideration. Perhaps further study of this subject will prove valuable in establishing the pathogenesis of certain poorly understood cutaneous processes. This group would include photosensitivity, pyoderma, hidradenitis suppurativa and lichen simplex chronicus but might embrace other unsuspected entities.

It is possible that any dye may prove injurious to a person sensitized to the dye in question. While certain ones, such as para reds and paraphenylenediamine, may have a higher index of sensitization,[18] the number of cases of dermatitis due to each dye is roughly proportional to the frequency with which clothes of each color are worn.

One of the most potent factors in the production of dye dermatitis is hyperhidrosis. Most of the incriminated dyes have been shown to be water soluble. When the material is placed in water, especially if warmed, the fluid becomes discolored with the dye. This is referred to as "bleeding." In most of my cases and in those reported in the literature "bleeding" was profuse. In 2 of my cases patch tests with the noxious material gave negative results after complete "bleeding."

Perspiration probably plays an important role in the causation of these dermatoses by "bleeding" the cloth. Many patients notice that they may wear the garment without trouble until warm weather or

18. Rostenberg, A., Jr., and Sulzberger, M. B.: Some Results of Patch Tests, Arch. Dermat. & Syph. **35**:433 (March) 1937.

exertion causes excessive perspiration. This factor has been emphasized by numerous previous authors. Patients may also complain of discoloration of the skin due to bleeding of the dye prior to the onset of the dermatitis.

To determine the frequency of "bleeding," three hundred and thirteen samples of new cloth were soaked in warm water. The amount of "bleeding" was classified as negative to 4 plus. Bleeding rated at 3 or 4 plus was noted in 110 cases (35.2 per cent). The percentage ranged from 7.7 per cent for gray cloth to 51.3 per cent for blue.

Most writers stress the frequency of dye dermatitis in women. In this series approximately 78 per cent occurred in men. Therefore, it is doubtful if sex or the menopause is of any significance in the development of sensitivity to dyes.

Many physicians feel that the patient with a dye dermatitis is polysensitive. F. A. Simon is the outstanding example of this. This polysensitivity may extend to other allergens beside dyes. However, it is equally true that the patient may be sensitive to a single dye only and may not react to closely related ones. Therefore, the patient may be able to wear a garment of a certain color but not another of the same hue.

SUMMARY

A review of the literature and a discussion of 8 cases of eruptions due to dyed clothing are presented.

Such lesions may be of industrial importance.

Dyed fabrics may cause dermatitis venenata, photosensitivity, pyoderma, hidradenitis suppurativa, lichen simplex chronicus and urticaria.

The importance of hyperidrosis and of "bleeding" is stressed.

The importance of sex and polysensitivity in the productions of these dermatoses is questioned.

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ACUTE ARTHRITIS ASSOCIATED WITH MYOCARDITIS SIMULATING ACUTE LUPUS ERYTHEMATOSUS

REPORT OF A CASE

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It has long been recognized that certain conditions of the skin are only a symptom of an underlying constitutional disturbance.

Osler [1] in his papers published between 1895 and 1904 on the visceral manifestations of the erythema group of cutaneous diseases presented a series of 29 cases. He stated the belief that they represented one family which was characterized by the similarity of the conditions under which they occurred and the frequency with which the various cutaneous lesions were substituted one for the other in the same patient at different times. In this group the cutaneous lesions are essentially only a symptom of a severe underlying disturbance.

Libman and Sacks [2] reported 5 cases of an unusual endocarditis, in 2 of which there were lesions resembling acute lupus erythematosus together with other erythematous eruptions. Their patients had had no previous organic symptoms. A summary of the observations were pericarditis, white centered petechiae, arthritis, erythematous and purpuric rashes, ulcerative lesions of the mucous membranes, pleuropulmonary symptoms, embolic phenomena, enlargement of the liver and spleen, acute glomerulonephritis, a tendency to leukopenia and repeatedly negative blood cultures. The authors stated the belief that the endocarditis was not an essential feature of the disease.

MacLeod [3] in his paper on lupus erythematosus expressed the opinion that the disease was not a pathologic entity due to a specific cause but was rather a peculiar cutaneous manifestation produced by any of a variety of toxic causes.

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1. Osler, W.: On the Visceral Manifestations of the Erythema Group of Skin Diseases, Am. J. M. Sc. **127**:1 (Jan.) 1904.

2. Libman, E., and Sacks, B.: A Hitherto Undescribed Form of Valvular and Mural Endocarditis, Arch. Int. Med. **33**:701 (June) 1924.

3. MacLeod, J. M. H.: Lupus Erythematosus, Arch. Dermat. & Syph. **9**:1 (Jan.) 1924.

In the case we are reporting, the articular and cardiac symptoms were the major part of the picture. The cutaneous manifestations developed late and appear to be of toxic origin, although they were clinically similar to lupus erythematosus disseminatus.

REPORT OF CASE

History.—L. F., a Caucasian man aged 33, a bookbinder, was admitted to St. Francis Hospital on Dec. 6, 1935, with the chief complaints of pain and stiffness of the joints, weakness, dyspnea on exertion and swelling of the ankles. He had been in good health until November 15, when he awoke in the morning with pain and stiffness in the knees, ankles and metacarpophalangeal joints. The involvement was fleeting, accompanied by pain, some swelling and redness. Not all the joints were involved at the same time. He also complained of pain in the right side of the chest aggravated by deep breathing. The symptoms persisted, and in about three weeks he began to suffer from dyspnea on exertion and swelling of the ankles.

The patient had had the usual childhood illnesses; otherwise his past medical and family histories were not significant.

Physical Examination.—At the time of admission the temperature was 99.2 F.; the pulse rate, 101; the respiratory rate, 28, and the weight, 155 pounds (70.3 Kg.). The eyegrounds were normal. The teeth had many fillings. The gums were in good condition. The tonsils were buried and cryptic. The lymph glands were not palpable. The heart sounds were distant and of poor quality, but no murmurs were heard. The blood pressure was 110 systolic and 90 diastolic. The heart was enlarged to the left, 4 inches (10 cm.) from the midsternal line. Examination of the chest showed diminution of expansion at the base of the right lung and impaired resonance with moderately coarse rales over this area. Whispered sounds were not transmitted. Abdominal examination showed the liver to be enlarged 2 inches (5 cm.) below the costal margin. The spleen was not palpable. The prostatic gland and its secretions were normal. Pitting edema of the ankles was present. There were stiffening of the metacarpophalangeal joints, with moderate swelling, and redness and tenderness over the right wrist, elbow and left knee. No petechiae or erythematous nodes were observed.

Laboratory Tests.—A roentgenogram of the chest on December 7 showed mottled clouding at the right costophrenic angle, probably bronchopneumonia, with adhesions on both sides and pleural thickening. A second roentgenogram on December 15 showed a similar picture with clearing in the bronchopneumonic area. The cardiac silhouette showed a definite enlargement to the left (fig. 1).

Repeated examinations (concentration method) of the sputum and cultures for tubercle bacilli gave negative results. The Wassermann and Kahn reactions on December 23 were negative. The urinalysis showed: specific gravity, 1.018; reaction, acid; no albumin, and no sugar. Chemical study of the blood on December 23 showed nonprotein nitrogen 31 mg. and sugar 103 mg. per hundred cubic centimeters. Blood taken for culture on December 23 was sterile after seventy-two hours. An agglutination test for Bacillus melitensis on the same day gave negative results. On December 6 examination of the blood showed 85 per cent hemoglobin and 5,040,000 red blood cells and 6,200 white blood cells per cubic millimeter, with a differential count of 77 per cent polymorphonuclears, 16 per cent small lymphocytes and 7 per cent large lymphocytes. Repeated urinalyses gave negative results. Roentgenograms of the teeth taken on June 8, 1935 showed apical abscesses in the teeth on the right side of the upper jaw. A right lower bicuspid was badly abscessed.

Course.—The patient improved with rest in bed. The infected teeth were extracted. His diet was high in vitamins and calories. He was given 10 grains (0.65 Gm.) of sodium salicylate every four hours with 5 grains (0.32 Gm.) of sodium bicarbonate. His condition improved; his joint symptoms quieted, and the dyspnea and swelling of his ankles disappeared.

During his stay in the hospital his temperature fluctuated between 97 and 99.4 F. in the afternoon. His cough completely disappeared, and he gained strength rapidly. He was discharged December 23, apparently in good condition, with the advice to rest at home for at least two months.

Subsequent Course.—In March 1936, while at home, the patient had a severe exacerbation of his joint symptoms affecting the knees, ankles and metatarsophalangeal and metacarpophalangeal joints. This involvement was accompanied by

Fig. 1.—Dec. 15, 1935. Pathologic changes in the chest, with enlarged cardiac silhouette.

slight swelling and fleeting tenderness. One week after this, his ankles began to swell, and he again became dyspneic on slightest exertion. He remained at home under treatment for the next three months, with no improvement. On June 4 he was readmitted to the St. Francis Hospital, with the same chief complaints.

Physical Examination.—There were no new significant findings. The condition in the chest had cleared, and his weight was 130 pounds (59 Kg.), his temperature 103 F., his pulse rate 120 and his respiratory rate 28. The blood pressure was 115 systolic and 90 diastolic.

Laboratory Tests.—The urine showed a specific gravity of 1.010, a heavy cloud of albumin, a few hyaline and granular casts and occasional red blood cells. On June 9 the nonprotein nitrogen was 41 mg. and the urea nitrogen 16 mg. per hundred cubic centimeters. Examination of the blood showed 80 per cent hemoglobin and 5,800,000 red blood cells and 5,800 white blood cells per cubic

millimeter, with a differential count of 60 per cent polymorphonuclears, 35 per cent small lymphocytes, 2 per cent large lymphocytes, 2 per cent eosinophils and 1 per cent basophils. Blood cultures made on June 9 and on June 30 were negative in seventy-two hours.

The sedimentation rate on June 30 was 75 per cent in the first thirty minutes and 80 per cent in one hour. Examination of the blood on July 13 revealed 70 per cent hemoglobin (Newcomber) and 3,200,000 red blood cells and 6,000 white blood cells per cubic millimeter, with a differential count of 65 per cent polymorphonuclears, 16 per cent small lymphocytes, 17 per cent large lymphocytes and 2 per cent monocytes. The basal metabolic rate was —4 per cent. Repeated examinations of the feces showed traces of occult blood. The Mantoux reaction was

Fig. 2.—Photomicrograph of tissue from a healing cutaneous lesion.

negative with 0.0005 mg. of purified protein derivative. Repeated urinalyses continued to show a heavy cloud of albumin, with many hyaline and granular casts and many red blood cells.

A histologic examination on September 21 of tissue from a healing cutaneous lesion showed thinning of the corium and flattening of the epithelial pegs. There was definite fibrous replacement of the elastic fibers, with perivascular infiltration of lymphocytes and occasional plasma cells (fig. 2).

The electrocardiogram taken September 26 showed regular sinus rhythm, diphasic T wave in lead I and depressed ST segment in leads II and III, with a left axis deviation.

Course.—Two days after admission, June 6, the patient had another acute exacerbation of the joint symptoms, during which his temperature rose to 104 F., and coincident with this an erythematous patch developed on the bridge of his nose. The eruption spread to the flush areas of the cheeks and produced a definite "butterfly" appearance. It was slightly elevated but not tender. During the course of three weeks the eruption spread to involve the lobes of the ears, the scalp,

the presternal region and the dorsa of the hands and arms. During the next month the mucous membrane of the palate, the entire face, shoulders and anterior aspects of the legs were involved. The eruption became deep red, and toward the edges of the lesion small gray scales appeared. On the cheeks the follicles were dilated and filled with corneous material. The eruption on the body, the

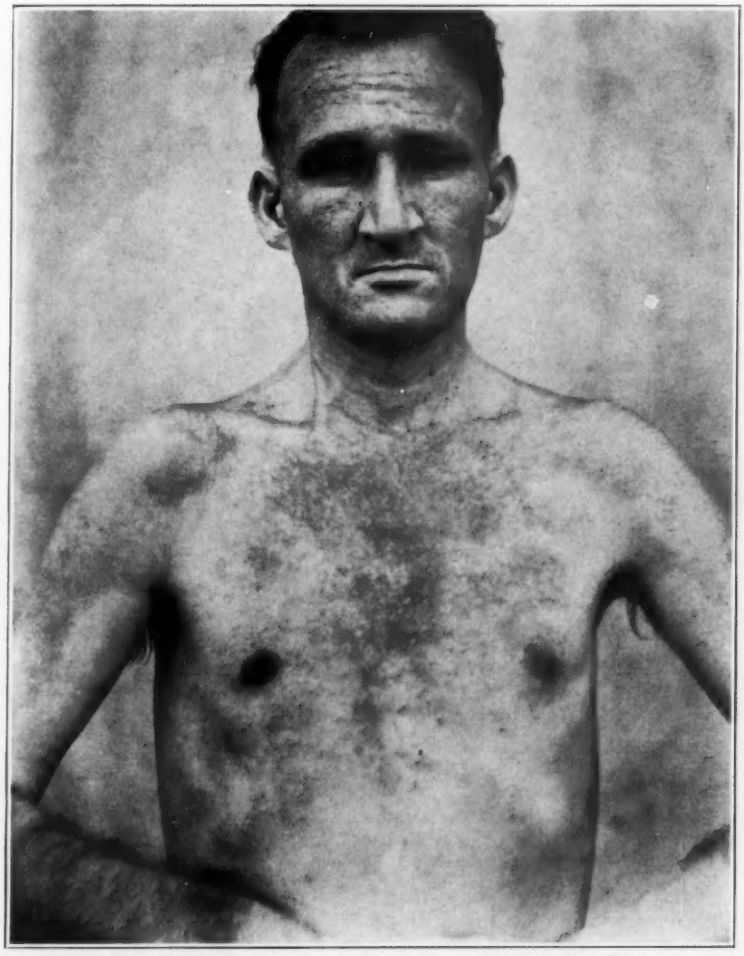

Fig. 3.—Distribution of lesions when eruption was subsiding.

hands and the feet consisted of small erythematous plaques, which is some instances became confluent. The lesions on the mucous membrane consisted of small bullae. From time to time new patches would appear as the older ones faded. The lesions caused only slight discomfort, principally itching and burning. The joint symptoms, however, became troublesome, and the swelling of the ankles increased. The prognosis looked hopeless.

Treatment.—Because of Wright's [4] successful treatment of lupus erythematosus with a gold compound and sodium thiosulfate and Forestier's [5] use of the drug for rheumatoid arthritis, it was felt that a therapeutic test might be made. Consequently starting July 10 he was given weekly intravenous injections of gold sodium thiosulfate, beginning with 5 mg. and increasing 2 mg. every week, until 25 mg. weekly had been reached. The dose was maintained at this level. There were no untoward reactions during the entire series, and definite improvement was noted. On August 11 he was given a transfusion of 500 cc. of citrated blood. In addition to this treatment, he was given pills of ferrous carbonate, 20 grains (1.3 Gm.) three times a day, and a high vitamin, high caloric diet, with cod liver oil, 2 drachms (7.4 cc.) twice a day. Codeine, 1 grain (0.065 Gm.), and A. S. A. compound (acetylsalicylic acid and acetophenetidin), 5 grains (0.32 Gm.), were used to control pain. His blood pressure during this time remained 140 systolic and 80 diastolic.

Fig. 4.—Aug. 19, 1939. Slightly enlarged cardiac silhouette. The pathologic changes in the chest have cleared, and no cardiac symptoms are present.

His response to treatment was excellent, and by October 1 his rash had almost completely subsided. His joint symptoms were improved, but he complained of severe pains in the lower part of the back. By November 14 he was afebrile. During his illness his temperature was septic, fluctuating between 99 and 102 F. The pulse rate followed a similar course. His urine still showed albumin and hyaline and granular casts but no red blood cells. The swelling of his ankles, although improved, still appeared after he stood for a few hours.

On December 10, during the patient's convalescence, a tonsillectomy was done under local anesthesia. A microscopic section of tissue showed chronic inflammation.

From Nov. 14, 1936, until Jan. 20, 1937, the date of his discharge from the hospital, his convalescence was uninterrupted. Gradually the edema of his ankles,

4. Wright, C. S.: Ten Years' Experience in the Treatment of Lupus Erythematosus with Gold Compounds, Arch. Dermat. & Syph. **33**:413 (March) 1936.

5. Forestier, J.: Rheumatoid Arthritis and Its Treatment by Gold Salts, Lancet **2**:646 (Sept. 22) 1934.

dyspnea on exertion, stiffness of his joints and cutaneous eruptions disappeared. His weight on Sept. 8, 1936, was 115 pounds (52.1 Kg.).

After his discharge from St. Francis Hospital his recovery was uneventful. He returned to work on July 5, 1937, and has been well ever since.

When he was examined on Aug. 19, 1939, a roentgenogram of his chest showed a slightly enlarged cardiac silhouette (fig. 4). The pleuropulmonary lesions had healed. An electrocardiogram showed a normal sinus rhythm, a left axis deviation and an inverted T wave in lead III. His blood pressure was 120 systolic and 80 diastolic, and his urine was normal. The skin and the joints appeared to be normal.

COMMENT

We realize the difficulty in trying to draw a conclusion from a single case, but there are certain factors which seem worth mentioning. The patient presented a bizarre rheumatic syndrome associated with a definite myocarditis for at least six months before the cutaneous manifestation appeared. He apparently was suffering from a severe constitutional disorder in which the dermatologic phenomena occurred as symptoms following an acute exacerbation of the systemic condition. In this respect the condition would fit into the group described by Osler. It is also analogous to that in the cases reported by Libman and Sacks. In their cases endocarditis was described, while in this case myocarditis was an outstanding observation.

The pathologic picture obtained from the biopsy of the skin certainly is not characteristic of any specific disease. This fact was emphasized by Satenstein,[6] who felt that there was nothing distinctive in the microscopic picture of lupus erythematosus disseminatus, since it merely showed an exudative inflammatory process. This has been our observation.

The question is: How is this condition to be classified? The majority of evidence points to a systemic disease in which the cutaneous manifestations are merely a symptom. If this is true, then it would fall in the group of diseases with erythema as presented by Osler.

SUMMARY

An acute systemic disease in a previously healthy adult, characterized by multiple joint symptoms, myocarditis, pleurisy, acute nephritis, enlargement of the liver and a cutaneous condition resembling acute lupus erythematosus disseminatus, which developed late in the illness, with recovery, is described.

The question arises whether this condition should be classified as an acute lupus erythematosus disseminatus or in Osler's toxic erythema group. We believe the latter classification is more appropriate.

6. Satenstein, D. L., in discussion on Goeckerman, W. H., and Montgomery, H.: Lupus Erythematosus: An Evaluation of Histopathologic Examinations, Arch. Dermat. & Syph. **25**:304 (Feb.) 1932.

CLAVICLE SIGN OF LATE CONGENITAL SYPHILIS

REVIEW OF LITERATURE AND REPORT OF SIX CASES

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In 1927 Higoumenakis presented before the Medical Society of Athens, Greece, a patient with congenital syphilis who showed tumefaction of the inner third of the right clavicle and a roentgenogram of the clavicle of another patient who, besides other stigmas of congenital syphilis, showed enlargement of the sternal extremity of the right clavicle. The author expressed his opinion, based on these observations, that the clavicle sign may be another stigma of congenital syphilis. He was able to verify this assumption by subsequent examination of all patients coming under his observation. The result of the investigation showed that: (1) the clavicle sign has a diagnostic value equal to other definite signs of congenital syphilis; (2) it occurs in greater frequency than the other stigmas of congenital syphilis and consequently is of greater diagnostic value, and (3) it is never encountered in persons with acquired syphilis or in healthy persons.

Since then, a few more patients have been shown to the Medical Society of Athens. It was in 1930 that Higoumenakis [1] reported a large series of cases (197) of congenital syphilis from the literature, with a description of 23 cases in which there were seven positive and sixteen negative Wassermann reactions. In the group with negative reactions, however, other stigmas of congenital syphilis were present. In this series of 197 cases, he found the clavicle sign present in 170 (86 per cent), on the right side in 157 and on the left side in 13. (The patients in whom the sign was on the left side were left-handed persons.) In 1937 the same author [2] reported 20 cases in which the sign was present, on the left side in 1 and on the right side in 19.

All the cases mentioned were observed in Greece. The numbers of cases reported in other countries are rather meager. In 1933 Touraine, Ribadeau-Dumas and Bernou [3] reported on a patient suspected

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1. Higoumenakis, G. C.: Deutsche Ztschr. f. Nervenh. **114**:288, 1930.

2. Higoumenakis, G. C.: Urol. & Cutan. Rev. **41**:799, 1937.

3. Touraine; Ribadeau-Dumas, C., and Bernou: Bull. Soc. franç. de dermat. et syph. **40**:120, 1933.

of having congenital syphilis on account of her dental stigmas. The inner third of her clavicle was enlarged, and a biopsy specimen taken from it showed periarteritis and endarteritis. However, the authors themselves did not attach any significance to this clavicular enlargement. Stokes [4] in 1934 stated that the sign was present in 4 per cent of 202 cases. In 1935 Dorne and Zakon [5] found it present in all of their 12 cases; Miskjian [6] presented 1 case before the Cleveland Medical Society, and Plishkin,[7] in Russia, reported some cases. In all these the sign was present on the right side. In the case reported in 1937 by Glickman and Minsky [8] the sign was on the left side.

This rather interesting new observation induced me to search for the alleged presence of the clavicle sign in patients with congenital syphilis coming to the dermatologic clinic of the National Medical College.

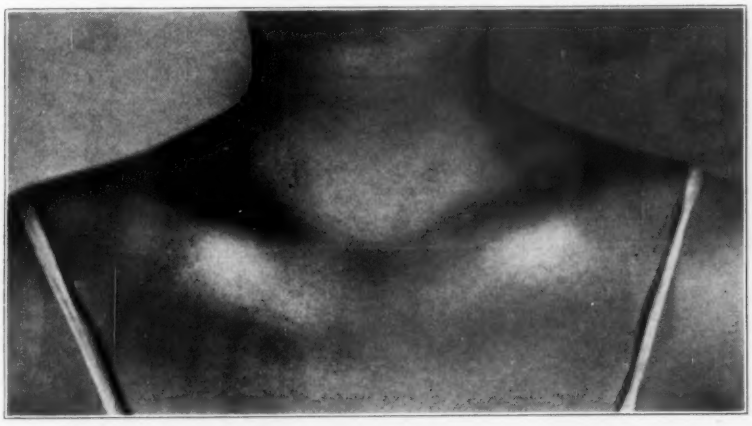

Fig 1 (case 1).—Clavicle sign in a woman aged 25.

Since May 1938, 6 patients with late congenital syphilis have come under my observation, and it is interesting to note that the clavicle sign was present in all of them.

REPORT OF CASES

CASE 1.—A woman aged 25 was admitted on Nov. 29, 1938 to the obstetric service of the First Hospital of the Red Cross Society of China, for her first

4. Stokes, J. H.: Modern Clinical Syphilology, ed. 2, Philadelphia, W. B. Saunders Company, 1934, p. 904, fig. 678 and p. 1255.

5. Dorne, M., and Zakon, S. T.: Enlargement of One Sterno-Clavicular Articulation as Valuable Clinical Sign of Late Prenatal (Congenital) Syphilis, Arch. Dermat. & Syph. **32**:602 (Oct.) 1935.

6. Miskjian, H. G.: Congenital Syphilis: Enlargement of the Sternal End of the Clavicle, Arch. Dermat. & Syph. **34**:701 (Oct.) 1936.

7. Plishkin, D. H.: Sovet. vestnik venerol. i dermat. **1**:16, 1935.

8. Glickman, L. G., and Minsky, A. A.: Radiology **28**:85, 1937.

parturition. Routine Wassermann, Kahn and Kline tests of the blood gave positive results. There was no history of abortion or miscarriage or of primary or secondary syphilis. The serologic tests of the cord blood of her baby and of the blood of her husband gave negative results. The medial third of her right clavicle was enlarged on inspection and palpation (fig. 1). Roentgenologic examination (posteroanterior view) revealed an increase of breadth of the inner third of the right clavicle in comparison with the left side.

CASE 2.—A woman aged 21 was first seen, on Nov. 24, 1938, in the surgical clinic, with the complaint of enlargement of the lower end of the right femur for five months. The condition was considered osteomyelitis. The Wassermann, Kahn and Kline reactions of the blood were positive. The sternal extremity of her right clavicle was enlarged. Roentgen examination confirmed this observation.

CASE 3.—A woman student aged 19 was first seen in the ophthalmologic clinic, on Sept. 5, 1938, because of reddening of the left eye of six months' duration. On examination the left eye showed chronic syphilitic interstitial keratitis. The Wassermann, Kahn and Kline reactions of the blood were positive. There was no other sign

Fig. 2 (case 5).—Clavicle sign in a woman aged 21.

of syphilitic infection. The inner third of her right clavicle was enlarged, as shown by physical and roentgen examinations. The condition responded to antisyphilitic treatment favorably and is now completely healed.

CASE 4.—A woman student aged 21 was observed to have repeated positive Wassermann, Kahn and Kline reactions on routine examination in May 1938. No other stigmas of congenital syphilis were present. The sternal third of her right clavicle was enlarged, as shown by inspection, palpation and roentgen examination. Roentgenograms showed that the enlargement was general but more marked in the medial half.

CASE 5.—A woman aged 21 was admitted on Jan. 23, 1939 to the medical service for pulmonary diseases. She was found to have lobar pneumonia. Routine blood examination disclosed positive Wassermann, Kahn and Kline reactions. The parents had a history of venereal disease, the nature of which was not known. General examination gave essentially negative results except that the medial third of the right clavicle was prominent on inspection (fig. 2). By palpation this prominence was verified, but the clavicle was narrower than the corresponding part on the left side. A roentgenogram also showed enlargement of the left clavicle.

CASE 6.—A woman aged 29 came to the dermatologic clinic on Aug. 15, 1939, for antisyphilitic treatment. The positive Wassermann, Kahn and Kline reactions of her blood were discovered when her baby was suspected of being syphilitic. She had been married for three years. Her general health was reported to have been excellent. She had never had any abortions or miscarriages and had a full term baby 2½ months old. On account of convulsions the baby was brought to the pediatric clinic, where, because of his general adenopathy, congenital syphilis was suspected. Then positive serologic reactions of both the baby and the mother (the patient) were disclosed. The serologic tests of her husband gave negative results. The patient's mother was reported to have had eight children. The first four were normal. The next three died either immediately or soon after birth. The mortality was attributed to the fact that the mother contracted syphilis from the father after the birth of the fourth child. The patient was the last child and was probably saved by treatment during her mother's last pregnancy. The

Fig. 3 (case 6).—Clavicle sign in a woman aged 29.

nature of the treatment was not known, as both parents had been dead a long time. The results of physical examination were irrelevant except that the sternal end of the patient's right clavicle was prominent on inspection and palpation (fig. 3).

CONTROL CASE.—A boy aged 10 years came to the ophthalmologic clinic on March 4, 1938, with the complaint of impairment of vision of the left eye for five months and blurring of vision of the right eye for ten days. The condition was found to be chronic syphilitic interstitial keratitis. The Wassermann, Kahn and Kline reactions of the blood were strongly positive. Both parents had positive serologic reactions. There were no other signs of congenital syphilis. Physical and roentgen examinations revealed only slight enlargement, if any, of the sternal end of the right clavicle.

COMMENT

According to Higoumenakis, the enlargement of the sternal extremity of the clavicle, as proved roentgenologically, is due neither to a defect in the statics of the patient nor to a loosening and distention of the articular capsule, but merely to an augmentation in the volume of the

bone caused by a hyperostosis resulting from a syphilitic osteitis. The enlargement usually occurred unilaterally on the right side except in left-handed persons, in whom it was on the left side. The enlargement may be explained as follows: Anatomically the inner third of the clavicle arises from a secondary nucleus which appears between the eighteenth and the twentieth year. The ossification of it is peculiar in that the bone is formed directly from connective tissue without preformation of cartilage, from a tissue, however, which is supposed to be infected with spirochetes in the syphilitic child. In early childhood the spirochetes remain latent locally. When puberty sets in, the increased activity of the person calls for an increased function of the clavicle during movement of the arm. The movement not only reactivates the spirochetes but also causes a constant friction of the medial extremity of the clavicle against the sternum, leading to hypertrophy of the sternal end of the clavicle: hence the clavicle sign.

All my 6 patients showed considerable enlargement of the medial extremity of the right clavicle by inspection, palpation and roentgenologic examination, with the exception of patient 5, in whom inspection revealed enlargement on the right side, but palpation and roentgenologic examination disclosed that, instead of the right, the left side was enlarged. Evidently the patient had bilateral enlargement. The enlargement on the right side was more anteroposterior, and that on the left, more vertical. Therefore, the former gave the prominence of the right clavicle on inspection and palpation, and the latter, the breadth of the left on palpation and roentgenologic examination. In the control case a slight enlargement was to be anticipated on account of the age. As is known, in a boy aged 10 years the secondary nucleus of the clavicle has not yet made its appearance, and the clavicle does not function so actively as during puberty; therefore, it is not well enough developed to be detected by examinations.

In order to rule out the possibility of leprosy as a cause of the sternoclavicular prominence, as observed by Nittis[9] in 1937, 6 young lepers, about the age of puberty, from a local leprosarium were examined both physically and roentgenologically, but the result was negative.

The observations in this small series of cases gives further confirmation to the constancy of the clavicle sign in late congenital syphilis. In 4 of 6 cases it was the only stigma of syphilis besides positive reactions to serologic tests. I fully agree with Higoumenakis that it is of high diagnostic value for late congenital syphilis. Furthermore, on account of its ready demonstrability, it is convenient and practical for physicians to keep it in mind in practice, as not infrequently the

9. Nittis, S.: Urol. & Cutan. Rev. **41**:625, 1937.

presence of this sign may serve as a guide in the diagnosis of certain conditions in which there are negative serologic reactions but other stigmas of congenital syphilis.

SUMMARY AND CONCLUSION

Six patients with late congenital syphilis were examined for the clavicle sign of Higoumenakis. In each there was an enlargement of the inner third of the right clavicle clinically and roentgenologically, and in 1 the enlargement was bilateral. The control patient, a boy aged 10 years with congenital syphilis, presented only slight clavicular enlargement, if any, as would be expected in consideration of his age. From this small number of cases, it appears that the sign is readily demonstrable; it is almost constantly present, and not infrequently it is the only presenting stigma; therefore, it is of great diagnostic value in late congenital syphilis.

ERYTHEMA EXSUDATIVUM MULTIFORME (HEBRA)

WITH CONJUNCTIVITIS AND STOMATITIS

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AND
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Erythema exsudativum multiforme with stomatitis and ocular involvement, though adequately described in ophthalmic literature, is of such rare occurrence that few clinicians are familiar with it. It seems worth while, therefore, to report a case, first, to give the disease wider clinical recognition and, second, to emphasize that there is a form of the disease in which the ocular involvement is of less serious consequence than that reported for the greater majority of cases recorded in American literature. Edmund,[1] in a comprehensive review of cases studied abroad and reported from Copenhagen in 1935, gave the impression that he was not familiar with the severer form of ocular complications recorded in this country. His impression was that serious ocular complications were rare and that when they did occur they were not part of the disease per se but were due to secondary or exogenous infection. That the ocular lesions are not of serious consequence appears to be a belief widely held abroad, for in the most recent French "Encyclopedia of Ophthalmology" [2] the statement is made that "The evolution of the conjunctival lesions is always satisfactory, the inflammation lasting two to three weeks." This view is in direct contrast to the experience and recorded observations of many ophthalmologists and others in this country. Stevens and Johnson,[3] Stark,[4] Wheeler,[5] Bailey,[6] Rutherford [7]

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1. Edmund, C.: Clinical Studies of Endogenous Conjunctiva Affections, Acta ophth., 1935, supp. 7, pp. 1-156.

2. Bailliart, P.; Coutela, C.; Redslob, E., and Velter, E.: Traite d'ophtalmologie, Paris, Masson & Cie, 1939, vol. 4, p. 552.

3. Stevens, A. M., and Johnson, F. C.: A New Eruptive Fever Associated with Stomatitis and Ophthalmia, Am. J. Dis. Child. **24**:526 (Dec.) 1922.

4. Stark, H. H.: Chronic Membranous Conjunctivitis of over Four Years' Duration, Am. J. Ophth. **1**:91, 1918.

5. Wheeler, J. M.: Destructive Purulent Ophthalmia Accompanying an Eruptive Fever with Stomatitis, Tr. Am. Acad. Ophth. **34**:179, 1929.

(Footnotes continued on next page)

and Ginandes [8] have reported partial and total loss of vision from this disease. There seems little doubt that a more severe form of ocular involvement not commonly seen on the Continent occurs in this country.

Beaudonnet,[9] who wrote an excellent monograph on this condition, credited Alibert and Bazin with having described the first cases ever to be recorded in 1822. One century later, in 1922, Stevens and Johnson called attention to the first cases in this country, believing that they were describing a new eruptive fever with stomatitis and conjunctivitis.

REPORT OF A CASE

R. B., aged 11 years, was admitted to the Brooklyn Hospital on Oct. 13, 1937. Ten days previously he had had a mild infection in the upper part of the respiratory tract, associated with a cough and slight fever. He had been given 15 grains (0.97 Gm.) of sulfanilamide. The cough and expectoration increased, and his temperature rose to 103 F. There were malaise, headache, fatigue and generalized weakness. He vomited several times. About six days after the onset, he became much worse and complained of extreme soreness in the mouth, with inability to swallow even liquids without severe pain. The breath was foul, having a heavy fetid odor.

He had had mumps and pertussis in early childhood. Each winter he had had a severe cold with bronchitis, leaving a cough which persisted for weeks. His tonsils and adenoids had been removed.

His family history was not significant. His home environment was excellent, and he had been reared in the best of surroundings.

Physical examination showed him to be acutely ill, abject in appearance, miserable and drooling freely. He coughed frequently and expectorated a great deal of viscid material. His cheeks had a dusky flush. His lips were somewhat cyanosed, swollen and covered with dark brownish crusts. Hemorrhagic spots were present on the oral aspect. His gums were swollen and hemorrhagic. A whitish gray membranous type of exudate was present on the mucosa of the greater part of the mouth, from which there was slight bleeding. The tongue was twice its normal size and heavily coated. There was moderate swelling of the cervical nodes, more particularly of the submaxillary group. There was edema of the conjunctivas, with marked injection of the vessels. Both eyelids were swollen, and a purulent discharge was present. A mucopurulent discharge was present in both nares. The pharynx was acutely injected and glazed, but no exudate was evident; and in general the throat did not appear to be involved to the same extent in the process that affected the buccal mucosa.

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6. Bailey, J. H.: Lesions of the Cornea and Conjunctiva in Erythema Exudativum Multiforme (Hebra): Report of Three Cases with Grave Ocular Sequelae, Arch. Ophth. **6**:362 (Sept.) 1931.

7. Rutherford, C. W.: Membranous Conjunctivitis with Loss of Eye Balls, J. A. M. A. **93**:1779 (Dec. 7) 1929.

8. Ginandes, G. J.: Eruptive Fever with Stomatitis and Ophthalmia: Atypical Erythema Exudativum Multiforme (Stevens-Johnson), Am. J. Dis. Child. **49**:1148 (May) 1935.

9. Beaudonnet, B.: Contribution à l'etude des manifestations oculaires de érythème polymorphe, Thesis, Paris, no. 354, Paris, Henri Jouve, 1894.

There was no impairment of resonance, but there were many scattered coarse moist rales in both lungs, more particularly at the base of the right lung posteriorly. The breath sounds were vesicular.

There was no cardiac enlargement. The sounds were of fair quality; the rhythm was regular, and there were no murmurs. The rate was 120 per minute.

The abdomen was soft and relaxed. The liver and spleen were not palpable. There was no edema of the extremities. No petechiae were present, and on admission no cutaneous lesions were evident anywhere on the body. The genitalia were normal. The reflexes were present and equal on both sides; the neck was not stiff, and the cranial nerves were normal.

Swelling of the eyelids, involvement of the lips and general appearance of dejection at the height of the disease.

A roentgenogram of the chest on admission showed only a moderate increase in root infiltration, with some extension along both lower bronchi.

The urine showed no sugar or albumin, but a trace of acetone was present. The hemoglobin was 85 per cent. The erythrocytes were 5,070,000 per cubic millimeter. The leukocytes totaled 22,600 per cubic millimeter, with 80 per cent polymorphonuclear neutrophils and 20 per cent lymphocytes. He was somewhat dehydrated, and perhaps the high red blood cell count and high hemoglobin content are thus explained.

A smear of the exudate present in the mouth revealed no Vincent organisms but a few pus cells, some short chain streptococci and some pneumococci. A blood culture was sterile.

For three days the picture remained unchanged, and the child was very ill, drooling profusely, coughing a great deal and unable to swallow without extreme pain owing to the condition in his mouth. On that day, one week after the onset of the stomatitis and conjunctivitis, typical erythema multiforme lesions of the herpes iris type developed on the extensor surface of both wrists, on the forearms and down both thighs and legs. A few scattered lesions were present on the abdomen. One lesion in particular, at the meatal orifice of the penis, caused great pain on voiding.

A direct blood transfusion of 300 cc. was given, followed by 100 cc. of 50 per cent dextrose solution in 120 cc. of physiologic solution of sodium chloride. He reacted well. The consultant from the ophthalmic service reported:

"The lids are not swollen. The margins of the lids are red. The conjunctivas of the lids and globe are intensely congested, giving a violet color, though there is no marked edema or swelling. The conjunctiva is covered with a thick, tenacious purulent secretion, which when removed does not show a membrane. There are a few papules on the under surface of the right upper lid. The corneas are clear and not involved. The fundi are normal."

On October 18 his condition was little changed. No new cutaneous lesions had appeared. A culture of material from one of the blebs on the skin showed a pure growth of Staphylococcus aureus. His cough was troublesome, accompanied at times by vomiting. The mouth seemed somewhat improved, though the mucosa remained deeply engorged, hemorrhagic and covered for the greater part by a whitish gray membrane. The roof of the mouth was deeply injected with many small petechiae scattered over the entire palate. A tenacious membrane was present on both lower lids. He was irritable, was difficult to manage and appeared to be uncomfortable. His temperature was 104 F.

On the next day he was less irritable, though the oral and cutaneous lesions were unchanged. Many coarse moist rales were present in both lungs, but no change in respiratory sounds was apparent. He evidently had a severe acute bronchitis. There was little purulent discharge from the eyes. A proprietary antacid and digestant powder was instilled in both eyes, and the membranes on both lower lids were rolled back. There was no bleeding.

On October 21, eight days after admission, his general condition was little changed. He was still bothered by a persistent productive cough, with mucopurulent expectoration. Voiding was painful and could be accomplished only after an ointment containing 1 per cent procaine hydrochloride was rubbed into the urethral orifice and a warm dressing soaked in boric acid solution was applied over the genital area. The membrane was still present on the right lower lid but not on the left. This was lifted off. There was not much purulent discharge.

On the next day his spirits were improved for the first time. His eyes were much better, and the purulent discharge was less thick, but the oral condition showed little improvement. His temperature was irregular and remained elevated.

On October 23 the red blood cell count was 4,740,000 per cubic millimeter. The hemoglobin was 79 per cent. The white blood cell count was 24,050 per cubic millimeter, with 80 per cent polymorphonuclear neutrophils, 18 per cent lymphocytes and 2 per cent endotheliocytes.

On October 25, twelve days after admission, the patient continued to have an irregular temperature up to 103 F., but his general condition was good. The oral lesions began to improve; swallowing was less painful, and the whitish membrane showed evidence of separating in a few areas, chiefly on the palate and on the inner aspect of both cheeks. The cutaneous lesions were drying more completely, the crusted blebs leaving a reddish but clean area where they had fallen.

Three days later the temperature was lower but still not normal, though his general condition was good. The chest was full of fine and coarse moist rales, mostly on the right side anteriorly and posteriorly and to a lesser extent over the lower lobe of the left lung posteriorly. An intradermal tuberculin test with 0.1 mg. showed a negative reaction. The oral lesions were much improved.

The eyes had continued to improve, the discharge gradually growing less. On this day both eyes were clear. There was no pus present. The local treatment with a 10 per cent solution of neo-silvol was discontinued, and only boric acid solution was used for occasional irrigation, with an ophthalmic ointment applied to the lids at night.

On October 30 the temperature was normal. The oral lesions had almost entirely healed, leaving a reddish but healthy-looking mucosa. There was a small patch of whitish exudate on the inner aspect of the lower lip. His eyes had healed almost completely, only slight injection of the conjunctivas remaining. The chest still showed evidence of bronchitis, with many coarse moist rales in both lungs.

On November 5, three weeks after admission, the patient was ready to be discharged. The eyes were entirely clear. The buccal mucosa was normal except for slight redness on the inner surface of both lips. His temperature was slightly above 100 F. at times, due, it was believed, to his persistent bronchial condition. Though most of the rales had cleared, some were still present at the base of both lungs. The lesions on the hands and feet still showed a reddish brown discoloration, though they were completely dry and healed

COMMENT

Erythema exsudativum multiforme with ocular involvement is a rare condition in this country and in western Europe, but it is frequently seen in Italy and in the Balkan states.[10] Rigler [11] described an outbreak in Turkey in 1852, and Dühring [12] reported an epidemic in the same country in 1896

The disease is usually sudden in onset, with fever, stomatitis, cutaneous eruption and conjunctivitis. It runs a protracted course, resulting often in partial or total loss of vision.

All degrees of ocular involvement may occur, from simple catarrhal conjunctivitis to phthisis bulbi with total blindness. Bailey and Duke-Elder [13] have both given excellent descriptions of the ocular lesions. It remains only to stress that corneal involvement is often insidious in onset and that in a large percentage of cases the condition ends with disturbed vision. Some of the reported phenomena leading to loss of vision have been (1) corneal ulcer with scar formation; (2) bilateral suppura-

10. Ocular Complications of Exudative Erythema, in Wood, C. A.: American Encyclopedia and Dictionary of Ophthalmology, Chicago, Cleveland Press, 1915, vol. 7, p. 4879.

11. Rigler, cited by Edmund.[1]

12. Dühring, E. Z.: Beiträge zur Lehre von den polymorphen Erythemen, Arch. f. Dermat. u. Syph. **35**:211, 1891.

13. Duke-Elder, W. S.: Text-Book of Ophthalmology, St. Louis, C. V. Mosby Company, 1938, vol. 2, p. 1734.

tive keratitis with perforation, resulting in phthisis bulbi and total blindness; (3) chemosis, subconjunctival hemorrhage, iridocyclitis, hazy corneas with vision limited to partial perception of light, and (4) pseudomembranous conjunctivitis with corneal involvement and symblepharon.

It is encouraging to note, however, that milder types of ocular lesions are compatible with severe involvement of the buccal mucous membranes. Catarrhal conjunctivitis with mild photophobia, lacrimation, itching and little discharge usually results in complete healing. Dühring, in a series of over 100 cases of the Hebra type of erythema multiforme, reported mild conjunctivitis only, from which there was complete recovery as the cutaneous and oral lesions cleared. Purulent conjunctivitis, though more often attended by serious corneal complication, may, as in our case, heal completely.

The severity of the involvement of the skin is not necessarily proportional to that of the buccal mucosa. The eruption may be mild, with the lesions well scattered, or it may be generalized and pronounced. Though the appearance of the cutaneous lesions precedes or occurs simultaneously with the onset of stomatitis and conjunctivitis, it may occur days later, as in our case. Herpes iris and bullous eruption are common types of lesions encountered, though any lesion described in the group of erythema multiforme may occur.

The cutaneous lesions have been described as maculopapular, occasionally vesicular and bullous. The eruption often appears progressively for a period of days or more, resolving, as a rule, with crusting and scaling and occasionally leaving brown pigmented areas, which finally disappear. Subjective symptoms of itching and smarting are usually absent. One lesion situated at the meatal orifice in our patient caused much difficulty in urination.

The condition in the mouth varies from simple stomatitis and gingivitis to a severe pseudomembranous inflammatory process, with great swelling of the mucous membranes of the tongue, lips, palate and cheek, making eating a serious problem because of pain in swallowing. Salivation may be extreme. The oral lesions are tender on manipulation; bleeding is variable, and, despite the usual methods of irrigation and oral hygiene, they are slow in resolving. Healing is usually complete.

The disease is generalized and systemic as well as local. Raffin [14] reported a case in which there was a streptococcic infection of the blood stream and a "bronchial catarrh." Hanke [15] reported a case, which terminated fatally, in which suppurative bronchopneumonia had been

14. Raffin, A.: Conjunctivitis, Rhinitis und Stomatitis aphthosa mit Erythema multiforme, Klin. Monatsbl. f. Augenh. **68**:216, 1922.

15. Hanke, V.: Der Herpes iris des Auges, Arch. f. Ophth. **52**:263, 1901.

present. Our patient had definite pulmonary involvement, manifested by a severe productive cough and expectoration of large amounts of watery, viscid material, unrelated to the salivation due to the oral lesions. His cough lasted many weeks after his temperature had returned to normal. In the last year repeated exacerbations of cough led to a study in which roentgenologic and physical findings all pointed to pulmonary fibrosis and early bronchiectasis.

The cause of this disease is not known. It is probably the result of systemic infection, though it has been attributed at times to ingestion of drugs or to toxic states. Because Vincent's organisms have occasionally been found in oral smears, the suggestion [16] has been recently made that these are perhaps the causative agents. It is much more likely that these organisms are secondary invaders unrelated to the underlying basic cause.

Once the disease is known its recognition should not be difficult. Briefly, it must be differentiated from septicemia, agranulocytosis, pemphigus, hemorrhagic measles, smallpox, Vincent's angina and syphilis. The usual means for excluding these need not be discussed here. The differentiation is either apparent or readily determined or becomes manifest as the illness progresses.

SUMMARY

A case of erythema exsudativum multiforme (Hebra) with stomatitis and conjunctivitis is reported. The prognosis of the ocular complications is discussed, and the possibility of a favorable outcome is indicated, even in the presence of a purulent conjunctivitis.

16. Chick, F. E., and Witzberger, C. M.: Erythema Exsudativum Multiforme Accompanying Oral Vincent's Infection, Am. J. Dis. Child. **55**:573 (March) 1938.

CASES OF FAMILIAL AND OF CONJUGAL PEMPHIGUS VULGARIS

USE OF TRANSFUSIONS OF BLOOD FROM CURED PATIENTS

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The term malignant in respect to pemphigus vulgaris is herein used to describe those types with which there is apparently going to be a fatal termination, as against the benign types from which recovery occurs. I am not in sympathy with the thought that when a patient with clinically clear pemphigus vulgaris recovers that patient did not really have pemphigus vulgaris. There are undoubted cases of clinically well marked pemphigus vulgaris in which a fatal prognosis has been given but, surprisingly, complete recovery occurs and there are no subsequent remissions.

So far as I have been able to ascertain, the only recorded instance in the American literature of familial malignant pemphigus vulgaris is that by Feldman,[1] who three years ago reported the disease in 2 brothers, both of whom died, 1 with pemphigus vulgaris of many months' duration and the other with pemphigus vulgaris of much shorter duration. In his textbook Sir Malcolm Morris [2] stated that Kaposi saw a patient with pemphigus vulgaris whose mother, sister and maternal uncle had been sufferers and several of whose children had it. Sir Malcolm himself had treated 3 members of one family for pemphigus vulgaris. Because of the recent observation of the disease in a father and son, the recording of this together with the report of pemphigus vulgaris in sisters and in a husband and his wife seen by me within the past eighteen years seemed of interest. The relation of the condition in the cases to be reported here to the benign types recently reported by the Haileys [3] is not clear except that the clinical picture in the present cases was altogether different and conformed entirely to the description of pemphigus vulgaris as found in standard textbooks.

1. Feldman, S.: Pemphigus in Brothers, Arch. Dermat. & Syph. **33**:730 (April) 1936.

2. Morris, M.: Diseases of the Skin, ed. 6, London, Cassell & Co., Ltd., 1917, p. 170.

3. Hailey, Howard, and Hailey, Hugh: Familial Benign Chronic Pemphigus, Arch. Dermat. & Syph. **39**:679 (April) 1939.

PEMPHIGUS VULGARIS IN FATHER AND SON

CASE 1.—The father, K. S., a white man aged 68, a meat and poultry retailer, was seen May 10, 1933, in consultation. Bullous lesions of the skin had developed about six weeks before I saw him. The clinical picture and a Nikolsky sign indicated pemphigus vulgaris. There was a history of nephritis three years previously. He was admitted to the Graduate Hospital of the University of Pennsylvania, where he died ten days later.

CASE 2.—The son, J. S., aged 55, a necktie manufacturer, was first seen Nov. 5, 1938, in the wards of Mount Sinai Hospital, in consultation. The patient had been well until July 1939, at which time a crusted lesion had appeared on his left cheek. The lesion was resistant to treatment, and in October nasal, oral and cutaneous lesions developed. A Nikolsky sign was present. A diagnosis of pemphigus vulgaris was made. In addition to general care, it was decided to give him blood transfusions, using blood, or serum if typing was different, from cured patients. Two patients cured of pemphigus vulgaris were obtained as blood donors; 1, a 68 year old patient of mine (cured in 1925 and seen by Dr. J. Frank Schamberg in consultation), supplied 250 cc. of blood which was given twelve days before the patient's death, and the other, a patient of Dr. J. V. Klauder's, supplied 500 cc. of blood, which was given seven days before death. Mercurochrome was given intravenously (1 per cent solution) in ascending daily doses (5 cc., 10 cc., and 15 cc.), the first dose six days before death, but toxic nephrosis developed subsequent to the 15 cc. dose. Pneumonia supervened two days later, and the patient died Dec. 9, 1939. Postmortem observations indicated that death was from extensive toxemia and bronchopneumonia and not from pemphigus as such. As a matter of fact, many of the lesions had healed, and others were slowly regressing.

CASE 3.—In another patient with pemphigus vulgaris under my care, first seen and hospitalized Nov. 23, 1939, a single transfusion of 500 cc. of blood from a third cured patient (originally seen by me but referred to the Philadelphia General Hospital and cured by Dr. John B. Ludy) given thirteen days before death was apparently of little value. Here again mercurochrome seven days before death was started in the same manner as in the second case, but toxic nephrosis developed with the third dose. There was no autopsy in this case, but death came suddenly and unexpectedly on December 9, apparently not from pemphigus, since a number of the cutaneous lesions had disappeared and others were regressing. The almost complete anuria attributable to the mercurochrome nephrosis was likewise improving.

It might be added that the second patient weighed 108 pounds (49 Kg.) and the third 224 pounds (101.6 Kg.), yet nephrosis developed in both with the same dose of mercurochrome. Perhaps much more blood from cured patients than was given to these patients is necessary before definite evaluation of this therapeutic procedure should be made. Judging from the literature of recent years, there are apparently so many cured patients (from the Davis treatment, the Engman tryparsamide method, mercurochrome, sulfonic acid compounds and germanin) that a fair trial of the suggested method for those with conditions that appear to be malignant should certainly be easy in such large centers as New York and Chicago.

PEMPHIGUS VULGARIS IN SISTERS

CASE 4.—Mrs. C. E., a white woman aged 62, a housewife, was first seen Sept. 3, 1931, with the history of the onset of her illness three months previously. The clinical picture was characteristic, and a Nikolsky sign was present. She was admitted to the Graduate Hospital of the University of Pennsylvania, service of Dr. J. Frank Schamberg, who saw her in consultation. She died November 22.

CASE 5.—Mrs. R. S., sister of Mrs. C. E., a housewife aged 43, was referred to me by Dr. William Ezickson in March 1936, and a diagnosis of pemphigus vulgaris was made. She was treated at the office and subsequently sent to the Graduate Hospital of the University of Pennsylvania (service of Dr. Fred Weidman), where she died suddenly on August 22, while quietly sitting in bed and while taking large doses (200,000 U. S. P. units daily) of vitamin D. Sudden deaths during the administration of and presumably from large doses of vitamin D have been reported.

I might add that Dr. John B. Ludy, who has successfully used this type of therapy, has in a recent personal communication informed me that he has observed several sudden deaths during massive vitamin D therapy, but a premonitory sign in patients receiving such large doses of vitamin D is the tendency to the immediate development of a striking pallor on turning from side to side or bending forward when in a sitting posture.

PEMPHIGUS VULGARIS IN HUSBAND AND WIFE

CASE 6.—Dr. J. S., a white man aged 53, a dentist, was seen by Dr. J. Frank Schamberg in 1923, and a diagnosis of pemphigus vulgaris was made. He lived seven months after the first lesion developed.

CASE 7.—In 1924, one year after her husband's death, the wife was referred to me. She had typical bullous lesions on the anterior part of the chest and on the left arm. A mild Nikolsky sign was elicited. From time to time over a period of five months, new bullous lesions developed here and there on the trunk. The lesions were typically those seen in patients with pemphigus vulgaris, but by the sixth month all lesions had disappeared. There were no subjective symptoms during the entire period. She remained well for over ten years, at the end of which time she succumbed to some other ailment.

320 South Eighteenth Street.

ERYTHEMA OF THE NINTH DAY FOLLOWING BISMUTH THERAPY FOR SYPHILIS

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All forms of untoward cutaneous manifestations may occur after the administration of a bismuth compound in the treatment of syphilis.[1] The commonest one closely simulates seborrheic eczema and consists of erythroderma which is pink to yellowish red and slightly raised, accompanied by fine dry scaling and a moderate to an extreme degree of pruritus. Persistence in the administration of the bismuth usually causes an extension of the eruption. Even cessation of its use is followed by a long period in which the eruption seems stationary. However, it fortunately does disappear and, in contradistinction to arsphenamine dermatitis, practically never eventuates fatally.

Recently Goldman and Clark[2] reported 2 cases, the first of their kind, of erythema of the ninth day following bismuth therapy. This dermatitis has been described in connection with arsphenamine therapy and differs in the essential mechanism of its production and in its course from the eruption that may occur as a sign of intolerance or hypersensitivity to an administered antisyphilitic arsenical. The rash is, as a rule, generalized, appears eight to ten (commonly nine) days after treatment, resembles either rubeola or scarlatina, is accompanied by fever, malaise and articular pains and disappears within two to five days after its onset. It does not as a rule reappear with further active antisyphilitic therapy. The condition in the following case illustrates an erythema of the ninth day associated with bismuth therapy.

REPORT OF A CASE

M. A., a woman aged 60 years, entered the medical outpatient clinic July 15, 1939, for treatment of severe pain about the right ear and on the right side of the face. Physical examination revealed aortic regurgitation and positive Wassermann, Kahn and Hinton reactions. Two additional examinations of the blood gave similar results. She was referred to the dermatologic clinic for treatment.

From the Department of Dermatology and Syphilology, Massachusetts Memorial Hospitals.

1. Grund, J. L.: Bismuth Eruptions, Boston M. & S. J. **196**:966 (June 16) 1927.

2. Goldman, L., and Clark, G. E.: Erythema of the Ninth Day Syndrome Associated with Bismuth Therapy, Am. J. Syph., Gonor. & Ven. Dis. **23**:224 (March) 1939.

On September 28 examination showed a group of typical syphilitic papules on the left buttock. The presence of a loud diastolic murmur over the precordium, Corrigan's pulse and capillary pulse, together with the active syphilitic eruption on her buttock, called for the employment of antisyphilitic therapy. On the same day she received 1 cc. of bismuth subsalicylate in oil intramuscularly; a saturated solution of potassium iodide orally was also prescribed. On October 9 and on October 11 she again received 1 cc. of the bismuth preparation. On October 9 she presented a burning generalized scarlatiniform eruption on her face, trunk, arms and legs. Late in the evening of October 6, after the second injection of bismuth and nine days after the first injection, she suddenly had chills, pains in the joints and a "burning" sensation over her entire body. She then noticed the aforementioned eruption. Calamine lotion was prescribed for the rash, which gradually disappeared five days after its onset. From October 11 to December 7 she received weekly injections of 1 cc. of bismuth subsalicylate in oil, without untoward effect.

COMMENT

This is the third case reported of ninth day erythema following bismuth therapy for syphilis. This instance illustrates the necessity for recognizing the true character of the entity. In cases in which active antisyphilitic therapy is required and in which it is inadvisable for various reasons to employ an arsenical, it is essential that it be kept in mind that even bismuth may be productive of a complication which at first glance calls for cessation of active treatment. Recognition of the comparatively benign nature and limited course of this particular cutaneous reaction undoubtedly enhances the success of treatment in the cases in which it occurs.

SUMMARY

A case of erythema of the ninth day associated with bismuth therapy is reported. Attention is called to the importance of its correct interpretation.

520 Beacon Street.

ACUTE SYPHILITIC TRANSVERSE MYELITIS

A CLINICAL STUDY AND REPORT OF A CASE

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In recent years there has been a decided lessening in the frequency of destructive lesions of the so-called tertiary stage of syphilis. This is also true of acute syphilitic transverse myelitis. Previous to 1912 numerous European observers had recorded a relatively high incidence of acute syphilitic paraplegia, over 200 cases, totaling from 17 to 26 per cent of all cases of spinal syphilis. However, in this country only 5 cases have been reported by four observers (Cole,[1] Skoog,[2] Lindemulder [3] and McCausland [4]) in the past fifteen years, though sporadic unreported cases have obviously occurred. In more striking contrast, Chung [5] has reported from China 34 cases of acute syphilitic paraplegia, or 39 per cent of the cases of spinal syphilis. This report comes from one clinic over only eight years in a country where syphilis is two to three times more prevalent than in this country. Chung's findings conform with those of other observers there. Although numerous speculations are offered for the difference, the final explanation will probably be made by a study in which the control and treatment of patients in China are comparable to standards in this country.

Acute syphilitic transverse myelitis affects an overwhelming predominance of males (about 10 to 1). There appears to be no relation between the site and severity of the primary lesion and the subsequent paraplegia. It is significant that practically all the patients in whom a syphilitic paraplegia developed had received no treatment or inadequate treatment. The duration of the syphilis in relation to the development of the myelitis varies from two months to over ten years, the latter condi-

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From St. Elizabeths Hospital, Washington, D. C.

1. Cole, H. N.: Acute Syphilitic Transverse Myelitis, Arch. Dermat. & Syph. **9**:102-113 (Jan.) 1924.

2. Skoog, A. L.: Case of Syphilitic Myelitis, J. Kansas M. Soc. **25**:339-340 (Oct.) 1925.

3. Lindemulder, F. G.: Myelitis Occurring in Primary and in Secondary Stage of Syphilis, Arch. Neurol. & Psychiat. **26**:167-169 (July) 1931.

4. McCausland, A. M.: Acute Syphilitic Transverse Myelitis, Am. J. Syph. & Neurol. **18**:216-220 (April) 1934.

5. Chung, M. F.: Thirty-Four Cases of Rapidly Developing Syphilitic Paraplegia, Arch. Dermat. & Syph. **14**:111-121 (Aug.) 1926.

tion usually occurring within one to three years after the primary lesion. Since there is a relatively short interval between the initial infection and the paralysis, it is to be expected that the incidence is greatest during the second and third decades of life. The usual precipitating factors described in other types of syphilis are offered here as being of etiologic importance, such as physical exhaustion, cold, alcohol, trauma and sexual excess, but their importance is rather dubious.

Examination of the spinal fluid shows that when invasion of the nervous system does occur it is extremely early in the course of the disease and that the nervous system may be seriously involved without symptoms. Also, from pathologic study, the syphilitic changes in the blood vessels and meninges are distributed over a much wider area than clinical symptoms suggest. In acute syphilitic transverse myelitis, the locus of diminished resistance appears to be the dorsolumbar region. Anatomically, this is the most dependent portion of the spinal fluid column and has the least blood supply. Considering its small diameter between the eighth thoracic and the first lumbar segment and the vital tracts contained therein, one or more lesions of the blood vessels here could easily produce a transverse myelitis.

The basic pathologic study shows that the vulnerable lesion consists of a thrombosis of one or more of the spinal vessels, usually arteries, such as the anterior spinal, the posterior spinal or the vasocoronal. The vessel most often affected is the anterior spinal artery.

Margulis [6] has offered a report and several interesting photomicrographic illustrations of 3 cases in which the condition was fatal. When the onset of the paraplegia is abrupt, a meningitic process may or may not be present with the thrombosis. The infiltration of the cellular elements into the wall of the blood vessel is often pronounced, though this need not necessarily be the case. The reaction is that of a panvasculitis, with infiltration of lymphocytes and plasma cells which results in endothelial swelling and proliferation, ending in a narrowing or obliteration of the lumen. During the course of reparation an attempt at recanalization through the thrombus may occur.

When the clinical course develops more gradually the meningitic reaction is a prominent underlying feature. There is a rich exudate of cells, predominantly lymphocytes, with endothelial and plasma cells. This is especially localized in and about the blood vessels on the periphery of the cord. There results perivasculitis, endarteritis and endophlebitis. The lumens of these small vessels are gradually encroached on, thereby interfering with the nutrition to the spinal cord. Less fre-

6. Margulis, M. S.: Pathologische Anatomie und Klinik der akuten thrombotischen Erweichungen bei spinaler Lues, Deutsche Ztschr. f. Nervenh. **113**: 113-145, 1930.

quently there may be an extension of the specific inflammation into the cord, usually invading the posterior and lateral columns.

Because of the thrombosis or narrowing of the spinal vessels, areas of ischemia develop within the spinal cord. The neural structures supplied by the affected vessels undergo softening and disintegration, with secondary ascending and descending degeneration of the spinal tracts.

Since the site and degree of the lesions of the cord are inconstant, the symptoms are manifold. In general it may be stated that the onset is more or less sudden. Depending on the rapidity with which the occlusion of the spinal vessels occurs, two groups may be distinguished. In the acute type of occlusion, the condition develops within twenty-four to forty-eight hours, with few or no premonitory signs. The patient suddenly presents complete flaccid paralysis of both lower extremities, with urinary retention and constipation. Associated sensory disturbances and trophic changes are invariably present. In the somewhat more progressive or subacute type there are prodromal symptoms, usually of meningeal irritation, extending over a period of several days to about four weeks. A constant severe headache not responding to ordinary sedation is a frequent complaint. Other subjective findings, named in the order of frequency in which they occur, are numbness of the lower extremities, weakness of the lower limbs, difficulty in urination or urinary retention, incontinence, constipation, backache and girdle sensation.

With the development of the paralysis, the neurologic changes depend on the degree of intensity of the morbid process in the different cord structures affected. In general, the deep reflexes of the lower extremities may be absent or increased. Sensation also varies and may be absent, diminished or dissociated. Sphincter control is lost slightly more often than it is spastic.

When the transverse lesion is complete there is total abolition of motion, usually with loss of the deep tendon reflexes and a complete sensory loss. Trophic changes are noted, and the sphincters are spastic. The picture may gradually change into one in which the deep reflexes are increased, a bilateral Babinski sign and ankle clonus are present and there is some return of sensation. There may be incontinence of urine and feces. On the other hand, in patients in whom the involvement is partial the paralysis may not be absolute; there are usually an increase of the deep tendon reflexes, a Babinski sign and clonus, with some hypertonicity of the muscles. The sensory function is not entirely destroyed, and symptoms of irritation, such as paresthesia, hyperesthesia or pain, are in evidence; or there may be a hypesthesia for all forms of sensation, as well as a sensory dissociation. Depending on the areas involved, the picture may also be purely motor or purely sensory.

A valuable aid in diagnosis is the history, for often the nature of the pathologic condition may be determined by the mode of onset alone. In a middle-aged person, syphilitic thrombosis of the spinal cord is a most important single factor in causing a sudden paraplegia. The diagnosis offers practically no difficulties when the condition develops soon after the primary lesion and when there is present a positive complement fixation of the spinal fluid, with an increase in protein and in cells, predominantly lymphocytes. This cellular response varies with the degree of accompanying meningeal irritation. There is always a disturbance of the colloidal gold curve, but the zone it is in should be interpreted cautiously and only in the light of existing symptoms. Otherwise it is diagnostic of nothing except an abnormality of the spinal fluid proteins. A negative result of a serologic test for syphilis may confuse the picture. However, this occurs fairly often; for example, in Chung's [5] 34 cases there were 4 in which the spinal fluids were normal, and in 2 of these, there were no serologic changes of the blood. In the case of Lindemulder [3] the Kahn reaction of the blood was strongly positive, but that of the spinal fluid was negative.

Differentiation must be made from such conditions as acute virus infections, which frequently cause transverse myelitis. Malignant growths, such as metastatic carcinoma, should be considered. I observed 2 cases of multiple myeloma in which there were no symptoms until acute paralysis developed; in 1 there was a paraplegia and in the other a tetraplegia. Benign tumors, syringomyelia, multiple sclerosis, tuberculosis (as in Pott's disease), embolic lesions and trauma with hematomyelia may also simulate a syphilitic paraplegia. In most of these the onset may be sudden, with the underlying pathologic condition a thrombosis or with occlusion of a spinal vessel as the basis of the symptoms.

As with practically all lesions of the nervous system, the prognosis for total recovery is guarded. The final result depends on several factors: the degree of destruction of the nerve tissue, the rapidity with which the circulation is reestablished, the resistance of the patient to complicating infections and adequate antisyphilitic therapy. In the 34 cases reported by Chung,[5] there were varying degrees of improvement in 21, a condition worse on discharge than on admission in 7, and death in 6, with a mortality of 17.7 per cent. In the 5 reported cases in the literature of this country during the last fifteen years, there was 1 death. At that, the mortality rate is not as high as it was a score or more years ago. In many of the cases the condition progresses to a chronic stage, in which the prolongation of life depends on maintaining the highest body hygiene. A few patients have recovered with few residuals of their illness. From a purely clinical standpoint, when the

signs and symptoms indicate a partial transverse involvement the prognosis is favorable; when the evidence is that of a total transverse lesion it is serious.

Opinions as to the therapeutic results are numerous and contradictory. Some observers have stated that antisyphilitic therapy was of little avail once the paraplegia developed; others, that it was the primary basis for improvement. Chung [5] expressed the opinion, "Recovery, if it occurs at all, will take place in spite of any treatment . . .," though he advocated vigorous antisyphilitic treatment to check any recurrence. This is true in some measure, but it does not altogether bear out the observation that prior to present therapeutic procedures the mortality rate was extremely high, whereas it has now been measurably reduced. Persistent and well regulated therapy should be carried on in the attempt to arrest the progress of the syphilis whenever lesions occur and to prevent further damage elsewhere. The management of each case is an individual problem. Before one institutes treatment, the possibility of further damage to already diseased vascular structures must be considered if trivalent arsenicals are first used. In general, it may be stated that patients do well with massive doses of iodides orally or intravenously. Heavy metals are primarily given, followed by the arsenicals.

REPORT OF CASE

J. M., a Negro barber aged 45, was admitted to a clinic about June 1, 1934. He complained of a "chest cold" and generalized muscular soreness. A Wassermann test of his blood had been made about every year, the last one being in 1932, when he had a small penile lesion. All gave negative results. After attending the clinic for two weeks, he mentioned the persistence of a penile "hair cut" obtained during the early part of April 1934. A Wassermann reaction of the blood proved positive. However, the spinal fluid was not examined. Intravenous arsenical therapy was immediately started. On the day of the second or third weekly injection the patient became dizzy and unsteady; a constant generalized headache developed, and hearing was practically lost in the right ear. Treatment continued for three more injections, when the clinic closed. His staggering became worse. The headache was so severe that he took a tablet of acetylsalicylic acid every hour, day and night, without relief. About August 1 his feet felt heavy, and a sensation of coldness developed in the lower extremities, extending upward to the anal region. He could hardly tell the position of his feet without observing them.

The patient was admitted to a municipal hospital on August 25. Neurologic examination revealed that the pupils were miotic and irregular. They reacted sluggishly to light and dilated in accommodation, particularly the right one. Speech was not impaired. The Romberg sign was present to a moderate degree. The upper extremities were normal. The knee jerks were normal or slightly diminished. Achilles reflexes were absent. Vibration and position sensation were normal. The patient walked with a wide ataxic gait. The diagnosis initially favored was that of atypical tabes dorsalis. The Wassermann reaction of the blood was strongly positive. Examination of the spinal fluid showed that the

Wassermann reaction was strongly positive; the protein content was 383 mg.; there were 715 cells per cubic millimeter, with 98 per cent lymphocytes and 2 per cent polymorphonuclear leukocytes, and the colloidal gold curve was 5555332100. On September 2, two days after the lumbar puncture, the headache cleared entirely, but agonizing pains developed, encircling the middle part of the abdomen. The legs became completely paralyzed, and a sensation of numbness developed. There was distention of the urinary bladder, with retention followed by urinary dribbling, and severe constipation. The patient was moved to the psychopathic department, where it was said that he had dementia. The diagnosis was changed to dementia paralytica, and on September 24 he was transferred to St. Elizabeths Hospital.

On admission the patient showed no evidence of an active psychosis. The physical examination revealed nothing significant other than the neurologic findings.

In October the patient was bedridden because of the paralysis of his lower extremities. The fundi were normal. The pupils were about equal, were slightly irregular and barely reacted to light, though they reacted well in accommodation. Hearing was diminished in the right ear, with air conduction greater than bone conduction. Bone conduction was reduced in the right as compared with that in the left ear. There was no defect in speech.

Sensory changes extended downward from the level of the fourth lumbar vertebra posteriorly and from 1 inch (2.5 cm.) above the symphysis pubis anteriorly to completely involve the lower extremities. Vibratory sensation was lost over the spinous processes below the fourth lumbar vertebra, over the pelvic bones, trochanters, malleoli and toes. Light touch was perceived, but the sensation was diminished. There was a decided hyperalgesia to pinprick. Differences of temperature were detected with difficulty. There was urinary frequency with burning. Constipation necessitated frequent enemas.

The abdominal and cremasteric reflexes were not elicited. The lower extremities showed a symmetric generalized atrophy. Muscle tonus was diminished. There was no active motion. The patellar and suprapatellar reflexes were active and equal. The ankle jerk was diminished on the left and absent on the right. Ankle clonus was present on the left. Signs of the Babinski group were not observed. On either side there was a pronounced flexion-withdrawal reflex.

The hemoglobin content of the blood was 80 per cent; the white blood cell count was 8,000 per cubic millimeter, the differential count being 41 per cent lymphocytes, 10 per cent monocytes, 47 per cent polymorphonuclear leukocytes, 1 per cent mast cells and 1 per cent band cells. The results of urinalysis were normal except for a faint trace of albumin and 15 to 20 white blood cells per high power field.

The Kolmer and Kahn reactions of the blood on September 25 were moderately positive. The condition of the spinal fluid on the same date was similar to that reported August 31.

Roentgenograms showed mild hypertrophic arthritis of the eleventh and twelfth thoracic vertebrae and of the first, fourth and fifth lumbar vertebrae.

In order to prevent therapeutic shock, the patient was given mercury inunctions, combined with a saturated solution of potassium iodide. The saturated solution of potassium iodide was continued, and the patient was given a course of neoarsphenamine. This was followed by intramuscular injections of bismuth subsalicylate.

On December 18 the Kolmer reaction of the spinal fluid was strongly positive; the protein content was 2 plus; there were 20 lymphocytes per cubic millimeter; a few red blood cells were present, and the colloidal gold curve was 5554322100. On March 19, 1935, examination of the spinal fluid showed a negative Kahn

reaction; the protein content was 2 plus; the cell count was 3 per cubic millimeter, and the colloidal gold curve was 4433210000.

By April there was subjective and objective improvement. The patient walked unaided, though the gait was somewhat unsteady. There was some weakness in the legs, especially affecting the left anterior femoral and hip muscles; occasional fibrillation of the muscles of the left leg occurred after he stood a long while. He had a sensation of automatic flexion and extension and of walking on a spring, especially in the morning. There was also a slight numbness of the legs, more in the left leg, but this sensation was gradually receding. He had some frequency of urination, with mild burning.

Nutrition and tonus of the lower extremities had almost returned to normal. Active motion was slow, with diminished force. Gait was on a slightly wide base. The deep reflexes were little changed. The various abnormal forms of sensation extending downward from the first dorsal segment underwent moderate improvement.

The patient continued to undergo hospital treatment with improvement until April 15, 1937, when he was discharged while on visit. His status on discharge was "not insane," and he showed a complete social recovery. It was indirectly heard that the patient had returned to his former occupation of barbering. The therapy given consisted of a total of 19.3 Gm. of neoarsphenamine and 3.63 Gm. of bismuth subsalicylate in oil, administered in alternating courses. At the same time, a saturated solution of potassium iodide was given during the early part of the treatment for a protracted period.

COMMENT

In this case there was a diffuse syphilitic meningeal inflammation which ushered in the prodromal symptoms three months after the primary infection. With it developed a syphilitic panvasculitis. This involvement was located particularly in the vicinity of the dependent portion of the spinal fluid column, that is, in the dorsolumbar region. Narrowing and thrombosis of the spinal vessels occurred, with subsequent paraplegia five months after the initial lesion. Clinically, the area most severely affected was the twelfth thoracic and first lumbar segment.

Treatment was instituted before the spinal fluid examination was made. As Wile and Marshall [7] and others have shown, early examination of the spinal fluid reveals that invasion of the nervous system, if it develops at all, will occur extremely early, in the first months of the infection in most instances. This involvement may often be serious but asymptomatic, thereby evading attention without a lumbar puncture. The therapeutic approach depends entirely on the results of the examination of the spinal fluid, and not until this is carried out can a case be managed with sagacity.

The posterior column signs and the first zone colloidal gold curve apparently influenced the initial impressions of tabes dorsalis and dementia paralytica in this case. Physicians have been prone to accept the belief that there are characteristic curves for tabes dorsalis, dementia

7. Wile, U. J., and Marshall, C. H.: A Study of the Spinal Fluid in 1,689 Cases of Syphilis in All Stages, Arch. Dermat. & Syph. **3**:272-278 (March) 1921.

paralytica and meningeal syphilis. Experience proves that only in syphilitic meningoencephalitis (dementia paralytica) with a history of inadequate treatment or none is a first zone (dementia paralytica) curve necessary. The converse is not true. Many other conditions have a first zone curve. Merritt and Moore,[8] for example, show the fallacy of designating the colloidal gold reaction on the basis of the pathologic changes. In a study of acute syphilitic meningitis there was a first zone (dementia paralytica) curve in 22 cases, a midzone (syphilis) curve in 28 cases and an end zone (meningitis) curve in only 3 cases. Reports on the colloidal gold curve in acute syphilitic transverse myelitis are meager. The cases of Skoog [2] and Lindemulder [3] each had an end zone curve. Chung [5] reported a gold curve in only 1 of his 34 cases, and this was in the end zone. The case of McCausland [4] and the case herein reported had first zone curves. Though the data are insufficient to draw definite conclusions, it appears that the syndrome of acute syphilitic transverse myelitis also does not have a characteristic colloidal gold response.

The clinical signs in this case were those of a partial transverse myelitis. This was an important factor in the prognosis and, in addition to the thorough antisyphilitic therapy, heightened the probability of a favorable outcome.

SUMMARY AND CONCLUSIONS

A clinical study of a patient with acute syphilitic transverse myelitis with paraplegia and his recovery are reported.

1. The pathologic process is fundamentally syphilitic panvasculitis leading ultimately to obliteration, by narrowing or thrombosis of spinal blood vessels.

2. Most patients receive inadequate treatment or none at all.

3. The condition occurs with greatest frequency within the first three years after the primary infection.

4. Tests of the blood and spinal fluid usually give positive reactions for syphilis. The protein content of the spinal fluid is elevated; there are an increased cell count and an alteration of the colloidal gold curve. However, the curve is not characteristic.

5. A valuable clinical prognostic guide is a differentiation between a partial and a complete transverse lesion. The prognosis is more favorable with the former type.

6. The results of treatment depend on the extent of injury to the nervous system, the rapidity with which the circulation is reestablished and, of primary importance, early, well regulated and sufficient antisyphilitic therapy.

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8. Merritt, H. H., and Moore, M.: Acute Syphilitic Meningitis, Medicine **14**:119-183 (Feb.) 1935.

THE SUDORIPAROUS GLANDS

III. SWEAT

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The function of the sudoriparous or coil glands has been the subject of much discussion and investigation since Simon [1] first noticed fat in sweat. Later Kölliker,[2] in 1853, expressed the view that the coil glands produce not only ordinary sweat but also a product rich in fats and albumin, with grades intermediate between the two extremes. Attention was called to the fact that the ordinary sweat glands, the axillary glands and the ceruminous glands constituted an unbroken order of development.

In 1857 Meissner [3] put forth the view that the sweat glands were the producers of fat, going farther than Krause and Kölliker in attributing to them the function of fat formation exclusively.

Next, in 1861, Henle [4] admitted that Meissner's arguments in proof of the formation of fat in the sweat glands were convincing but adopted a more moderate view and concluded that the sweat glands as a whole secrete fat and, besides that, at times watery sweat without fat. This view was likewise held by Unna [5] and Duhring.[6]

As the result of an interesting investigation, Unna [7] found that under normal conditions the sweat glands of the soles always contain fat

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1. Simon, F., cited by Unna.[7]

2. von Kölliker, A.: Manual of Human Microscopic Anatomy, translated by G. Busk and T. Huxley, Philadelphia, Lippincott, Grambo & Co., 1854, p. 202.

3. Meissner, G.: Zur Funktion der Knaueldrüsen, in Henle, J., and Meissner, G.: Bericht über die Fortschritte der Anatomie und Physiologie im Jahre 1856. Als besondere Abteilung der Zeitschrift fur rationelle Medizin, Leipzig and Heidelberg, 1857; in Unna, P. G.: Dermatologische Studien, Leipzig, L. Voss, 1889, no. 9, p. 145.

4. Henle, J.: Handbuch der systematischen Anatomie des Menschen, Brunswick, Friedrich Vieweg u. Sohn, 1866, vol. 2, p. 29.

5. Unna, P. G.: The Function of Sweat Glands in Man, Brit. J. Dermat. **6**:257, 1894.

6. Duhring, L.: Cutaneous Medicine, Philadelphia, J. B. Lippincott Company, 1895, pt. 1, p. 62.

7. Unna, P. G.: Der Nachweis des Fettes in der Haut durch sekundäre Osmierung, Monatsh. f. prakt. Dermat. **26**:601 (June) 1898.

globules in different quantities. Later he concluded that the ordinary sweat glands in man secrete fat which, though similar to that of the sebaceous glands, is different from it, as stearic and margaric acid differ from oleic acid.

SWEAT

A product of the secretion of sweat glands is poured out on the surface of the skin. Whether it seeps out as insensible perspiration or comes out directly owing to the influence of high temperature, this secretory product presents certain important characteristics.

Under the name of sweat the secretions and excretions of the glands must be considered together, for it is difficult to draw any hard and fast distinction between the two. This becomes apparent when one remembers that a secretion is a product which is of further use to the organism. Although an excretion is a waste product, it may be of some indirect use during the process of its discharge from the body. The products of the different cutaneous glands cannot readily be separated from one another or even from the products of the epidermic cells which surround their ducts.

Secretions from the apocrine and eccrine glands maintain the skin in a supple condition and preserve the acuteness of its tactile sensibility. Another purpose served by the discharge of sweat is the removal of waste products formed by the body. Its cleansing effect is also remarkable, and many a laborer who never takes a bath and rarely washes more than the extremities of his body is kept clean and healthy owing to the fact that he works hard enough at times to sweat profusely.

Pembrey [8] stated that it would be more desirable if there were more sweating from healthy muscular work and less washing with hot water and soap, because the natural fatty products of the skin and its glands protect the body from micro-organisms.

Physical Properties.—Normally sweat is a colorless liquid, being clear when freed by filtration of the epithelium and fat present. The odor is characteristic, although subject to slight variation, depending on the part of the body it comes from. It has a salty or, occasionally, a bitter taste. The specific gravity is about 1.005 but may vary, being greater at the beginning of sweating. The acid reaction of sweat is due to the presence of fatty acids or acid phosphates of sodium and potassium which it contains. An adult secretes 700 to 900 Gm. of sweat daily. Under the influence of heat, exercise or disease this quantity may be greater than 2 liters in twenty-four hours.

Chemical Properties.—Sweat is made up of a dilute solution of mineral salts, of which sodium chloride is the most important. Present

8. Pembrey, M. S.: The Functions of the Skin, Brit. J. Dermat. **22**:225, 1910.

also are potassium chloride, alkaline salts, organic acids, some urea, a slight amount of fatty matter, odoriferous substances, volatile fatty acids, formic acid, acetic acid, proprionic acid and butyric acid. It is the volatile bases (trimethylamine and methylamine) and the fatty acids (butyric acid, caproic ethers and valeric ethers) which give to the sweat its fetid odor.

Favre [9] gave the chemical quantitative analysis of 1,000 cc. of sweat as follows: sodium chloride, 2.230 Gm.; potassium chloride, 0.244 Gm.; alkaline sulfates, 0.012 Gm.; alkaline phosphates, traces; albuminates, 0.005 Gm.; earthly phosphates, traces; alkaline lactates, 0.317 Gm.; alkaline sudorates, 1.562 Gm.; urea, 0.043 Gm.; fatty matter, 0.014 Gm.; epithelium, 0.043 Gm., and water, 995.573 cc.

Excretory Capacity.—Substances similar to those found in the urine, such as urea, ammonia, uric acid, creatinine, chlorides, phosphates, sulfates and certain enzymes, also occur in the sweat. The quantity, however, is normally far less.

Cornbleet,[10] investigating the excretion of bromine through the skin, found that the sweat glands have a limited capacity for secreting it. Shortly after the continuous ingestion of bromides the concentration of bromine in the sweat reaches an upper limit which is maintained for a long time. The epithelium also may contain large amounts of bromine during the accumulation of bromides in the body with continuous ingestion. Bromine was also found to be a normal constituent of sweat and of the exfoliated epidermal cells.

Bismuth, on the other hand, was not found in the sweat of 4 patients studied by Schaffer.[11] Various preparations were used and given intramuscularly twice weekly in doses of 0.03 Gm. The time between the last dose of a bismuth compound and the collection of sweat varied from one hour to three days. It is of interest to note that urine from these same subjects showed the presence of bismuth in amounts of from 0.6 to 1.5 mg. per hundred cubic centimeters.

The low excretion of sulfanilamide in the urine of Highman's [12] patients during extremely warm weather led to its being found in appreciable quantities in the sweat.

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9. Favre, A., cited by Gautier, A.: Leçons de chimie biologique normale et pathologique, ed. 10, Paris, Masson & Cie, 1897, p. 446.

10. Cornbleet, T.: Excretion of Bromine Through Skin, J. Invest. Dermat. **1**:399 (Dec.) 1938.

11. Schaffer, H. W.: Studies of Perspiration: I. Bismuth, Arch. Dermat. & Syph. **22**:624 (Oct.) 1930.

12. Highman, W. J.: Excretion of Sulphanilamide in Perspiration, J. Lab. & Clin. Med. **23**:790 (May) 1938.

Stolyhwo,[13] after a series of experiments, concluded that toxic substances capable of eliciting the Shwartzman phenomenon may be demonstrated in the urine and sweat of human beings with typhoid fever. These toxic substances are apparently excreted in amounts sufficient for elicitation of the phenomenon during the period of severe toxic manifestations.

Uric acid and diastase are apparently constant constituents of normal sweat. Antitoxin is more frequent in the sweat than in the urine of people with normal renal permeability. The traces of various organic substances found in sweat are commonly considered to have been obtained from the blood. The same is probably true for the antitoxin in the sweat of people who have high concentrations of the immune substances in their blood. Neill,[14] in discussing the loss of immune substances from the body, stated that significant amounts of diphtheria antitoxin were found in the sweat of 3 men possessing a large amount in their blood (2 with natural immunity and 1 previously injected with toxoid); none, however, was found in the sweat of people with negligible amounts of antitoxin in their blood.

The various vitamins are undoubtedly present in sweat, and this impression has been confirmed by Lillienfeld, Wright and MacLenathen,[15] who found appreciable amounts of ascorbic acid (vitamin C).

Mercury seems to be readily excreted through the skin, but the commoner dyestuffs used for colorimetric estimation of renal function are not.

The sweat glands have the power of concentrating ammonia, and the ratio of ammonia nitrogen to total nitrogen in the sweat is considerably higher than that in the urine.

The secretion of perspiration is also modified by changes in the activity of other secretions, particularly those of the lungs and kidneys, whose function is frequently vicarious with that of the skin and vice versa. Thus during the summer and in warm climates the perspiratory secretion is augmented, while the exhalation from the lungs and the quantity of urine are diminished. In the winter and in the colder climates the reverse is true.

Lining,[16] an American, as early as 1740 made some interesting experiments on the relative quantities of perspiration and urine during

13. Stolyhwo, N.: Toxic Substances in Sweat of Typhoid Fever Patients as Demonstrated by the Shwartzman Phenomenon, J. Immunol. **30**:235 (March) 1936.

14. Neill, J. M.: Loss of Immune Substances from Body: Diphtheria Toxin in Human Sweat, J. Immunol. **21**:101 (Aug.) 1931.

15. Lillienfeld, A.; Wright, I. S., and MacLenathen, E.: Intramuscular Injection of Ascorbic (Cevitamic Acid) and Excretion in Sweat, Proc. Soc. Exper. Biol. & Med. **35**:184 (Oct.) 1936.

16. Bing, F. C.: John Lining, an Early American Scientist, Scient. Monthly **26**:249, 1928. Lining, J.: Metabolism Experiments, Phil. Tr. Roy. Soc., London **42**:49, 1743; **43**:318, 1745.

the warmer and colder months of the year. He weighed himself each morning immediately after rising and again at night just before going to bed. The balance used is not described, but according to the data reported, it weighed in pounds, ounces and drachms. The results obtained, although crude, gave a fairly accurate comparison.

Month	Perspiration	Urine
July	86.41	43.77
May	68.11	56.15
October	40.78	46.67
February	37.45	77.86

His conclusions were as follows:

> The volume of urine is roughly inversely proportional to the amount of perspiration; the amount of perspiration being greatest in the hot months of summer and least in the winter and the volume of the kidney secretion being conversely greatest in winter and least in summer.

An example of the effect of heat may be observed after leaving a warm apartment, especially after indulging in stimulants, when as a result of exposure to cold air a sudden check is given to the cutaneous function, while that of the kidneys is suddenly and actively called into exercise.

The rate of ventilation has a decided effect on the amount of water vapor given off by the skin. The amount increases with the rate of ventilation up to a certain point at which it seems that a cooling effect prevents a further increase.

Cotton garments do not seem to offer any obstacle to the loss of water vapor from the skin, at least not when they have been in contact with the skin a number of hours.

In the case of the infant, such factors as fretfulness, crying and a rise of room temperature increase the insensible perspiration. The term insensible perspiration is believed to have been first used by Sanctorius,[17] an Italian physician, as early as 1614. By "perspiratio insensibilis" is commonly meant (Benedict and Root[18]) those gaseous emanations from the body which do not appear in the form of sensible moisture or sweat, in other words, the insensible, intangible but weighable gaseous and vapor productions arising from the lungs in the process of exhalation and from the skin by due process of vaporization, i. e., excretion of moisture and carbon dioxide.

Isenschmid[19] in 1918 pointed out that the insensible perspiration is equal to the weight of the water given off, minus the weight of the

17. Sanctorius, S.: Ars de statica medicina, Venice, M. A. Brogiollus, 1614.

18. Benedict, F. G., and Root, H. F.: Insensible Perspiration, Arch. Int. Med. **38**:1 (July) 1926.

19. Isenschmid, R.: Die Bestimmung der Wasserbilanz am Krankenbett, Med. Klin. **14**:1128, 1918.

carbon dioxide given off, minus the weight of the oxygen consumed. From the diet one can compute the amount of carbon dioxide produced and the oxygen required to oxidize the food. Hence, if the subject is in metabolic equilibrium, one can compute in this way the insensible water loss. Observations by Loewy [20] of persons whose skin was devoid of sweat glands seem to demonstrate that insensible perspiration is entirely independent of the sweat glands.

Anhidrosis.—Nothing demonstrates more clearly the heat regulation function of the sweat glands than their failure to produce sweat when the body temperature becomes increased.

Zuntz and Tendlau [21] made observations on a man whose skin was devoid of sweat glands. His temperature rose to 39 C. (102.2 F.) when he was exposed to the heat of the sun in summer or when he did muscular work over a long period. The blood vessels of the skin were dilated in a normal manner, but radiation and conduction were not sufficient to prevent his temperature from rising while working. He overcame this serious handicap caused by the absence of sweat by frequently soaking his shirt in water and allowing its evaporation to cool his skin.

Anhidrosis resulting from a toxic dermatitis has been noted by Patzschke and Plaut.[22] This dermatitis developed in a patient after she had been working on and wearing furs that had been treated with naphthalene. The functioning of the sweat glands was completely abolished thereafter, and a high fever developed in the young woman at work and when exercising or dancing. This generalized anhidrosis remained stationary for one year before an attempt was made to stimulate the sweat glands. As a result of minute subcutaneous injections of pilocarpine hydrochloride followed by ultraviolet irradiation, improvement rapidly occurred. Such minute doses as were used (0.006 Gm.) are not supposed to influence normal skin. The arrest of perspiration in a patient with a cutaneous disease is often attended with congestion of the mucous membranes.

Another patient, reported on by Richardson,[23] in whom the absence of sweat glands had been demonstrated by microscopic examination, was carefully studied, and the following observations were made. A normal quantity of water vapor was liberated when at rest, and 30 per

20. Loewy, A.: Untersuchungen über die physikalische Hautwasserabgabe, Biochem. Ztschr. **67**:243, 1914.

21. Zuntz, N., and Tendlau, B., cited by Pembrey, M. S.: The Functions of the Skin, Brit. J. Dermat. **22**:287, 1910.

22. Patzschke, W., and Plaut, R.: Ueber einen Fall von allgemeiner Anhidrosis nach toxischer Dermatitis, München. med. Wchnschr. **68**:1117 (Sept. 2) 1921.

23. Richardson, H. B.: Clinical Calorimetry: Effect of Absence of Sweat Glands on Elimination of Water from Skin and Lungs, J. Biol. Chem. **67**:397 (Feb.) 1926.

cent of this came from the lungs, while the remainder was from the skin. Vaporization of water from the skin thus occurred independently of the sweat glands. The quantity of water eliminated was sufficient in combination with the heat loss in other ways to keep the body temperature within safe limits, even with exercise which increased the metabolism for thirty-five minutes to two and a half times the resting value. During this exercise the elimination of water did not increase as in a normal person, and the body temperature rose somewhat higher. Also with exposure to external heat there was no increase in the quantity of water vaporized, and the body temperature rose sharply.

A study of these 2 cases tends to confirm the theory that the sweat glands except under abnormal conditions are not a vital necessity in the heat regulation of the body.

According to Chevens,[24] a congenitally malformed skin, such as is described under the name of xeroderma, is one in which there is definite underdevelopment in the number of sweat glands, combined with hyperkeratosis, which further reduces their output. It is possible, therefore, that lack of capacity for sweating may be due to a reduced number of glands. The delay of sweating may also be due to the underdevelopment or structural deficiency in the nervous system.

Hyperhidrosis.—Hyperhidrosis is a functional disorder of the sweat glands characterized by an excessive secretion of sweat. The condition is further classified as hyperhidrosis generalis, localis, universalis, paradoxica and unilateralis.

A. Hyperhidrosis generalis signifies that most of the body may be involved, but normal areas may be observed between the affected ones. This condition is often symptomatic and is seen with malarial fever, tuberculosis and typhoid fever.

B. Examples of localized sweating are familiar to us all, occurring on the palms, soles, forehead, eyelids, nose, cheeks, axillas or genitalia.

C. Hyperhidrosis universalis is a term used to describe involvement of the entire body surface.

D. Hyperhidrosis paradoxica may occur with fever or a subnormal temperature. It is also observed with nervous conditions and lesions of the spinal cord.

E. Hyperhidrosis unilateralis when present should excite suspicion of a disease of the sympathetic system. It occurs frequently with pneumonia and may be found with various conditions dependent on reflex origin from the pneumogastric nerve, aneurysm, hemiplegia of cerebral origin and peripheral facial paralysis.

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24. Chevens, F.: Sudorific Reaction, J. Ment. Sc. **72**:331 (July) 1926.

Pieri [25] classified hyperhidrosis etiologically under three headings: 1. The first type he referred to as sympathetic hyperhidrosis; this occurs secondary to involvement of other organs and results from dysfunction of the nervous system. 2. The second type he referred to as functional hyperhidrosis; it is an exaggeration of the normal sweating function. 3. The third type he called idiopathic hyperhidrosis; it included examples of hyperhidrosis the origin of which he had been unable to establish.

Circumscribed hyperhidrosis as described by Stillians [26] is a common disorder of the skin which may usually be classed among the petty annoyances of life. When accompanied by bromidrosis, dyshidrosis or hyperkeratosis it often assumes a much more important role, incapacitating the person for work or pleasure. Strong body odors cause a person to avoid contact with others.

Roberts,[27] in an ably presented review of a case of hyperhidrosis and cyanosis in which the condition was relieved by sympathectomy, stated that the responsibility for the morbid condition lay not in the central nervous system but in the mesenchymal cells that surrounded the capillaries of the cutis. It was his opinion that this control is physical or physicochemical rather than nervous and that the nerve impulse is merely the trigger that starts the dermal reactions.

TESTS FOR THE PRESENCE OF SWEAT

Minot's test accurately reveals the presence of moisture and is therefore useful in determining the distribution of sweat and also lends itself to photography. It consists of the iodine-starch color reaction which takes place in the presence of moisture.

Another method is to stripe a patient's skin with a saturated alcoholic solution of cobaltous chloride before exposing to dry heat. The saturated alcoholic solution of cobaltous chloride, which is deep blue, changes to red on the addition of water, and where sweating occurs the blue stripes at once become a deep pink. This is, according to Berkman and Horton,[28] an excellent clinical test for determining the presence of sweat.

PHYSIOLOGY OF SWEATING

When the body is exposed to a raised temperature a series of physiologic changes takes place, the purpose of which is to retain the blood heat at "normal level."

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25. Pieri, G.: Contributi alla chirurgia del sistema nervoso vegetativo; la cura della iperidrosi, Arch. ital. di chir. **31**:117 (Feb.) 1932.

26. Stillians, A. W.: Hyperhidrosis, J. A. M. A. **67**:2015 (Dec. 30) 1916.

27. Roberts, H. L.: The Removal of the Inferior Cervical Ganglia and Its Effect on Hyperhidrosis and Cyanosis of the Hands and Feet, Brit. J. Dermat. **46**:126 (March) 1934.

28. Berkman, J. M., and Horton, B. T.: Hysteria Associated with Absence of Sweating, Proc. Staff Meet., Mayo Clin. **12**:168 (March 17) 1937.

The temperature of the skin is usually at a somewhat lower level than that of the blood. During life there is a constant dissipation of heat from the body, partly by radiation and convection and partly by insensible perspiration. If the heat production is increased there is a general superficial vascular dilatation and in consequence a greater surface of blood vessels exposed to the cooling effect of the surrounding medium. Should this surrounding medium be at too high a temperature to allow sufficient loss of body heat, there is a decided increase in the moisture output of the skin, and cooling is provided by a greater evaporation. It is thus clear that dry heat is more easily borne than moist heat, because with moist heat evaporation can take place only with difficulty.

Although physiologic experiments lead one to believe that the sweat glands are innervated by the autonomic system, results obtained by pharmacologic methods are not such as would be expected. Epinephrine, which stimulates the sympathetic system, does not promote sudoriparous activity but, according to Langley,[29] lessens the production of sweat by its vasoconstrictor action. Sweating time is usually delayed in persons that possess a much impaired peripheral circulation.

It is also conceivable that lack of or delay in sudoriparous activity may be due to a defect either in the glands themselves or in the nervous mechanism by which their activity is brought about.

A far greater percentage of patients under treatment for mental diseases show a wide variation from the normal mean.

FACTORS INFLUENCING HYPERHIDROSIS

The quantity of perspiration is altered by a variety of circumstances which affect the body physically or through the agency of the nervous system. The temperature, current hydrometric condition of the atmosphere and stimulation of the skin belong to the former, while excited or depressed nervous powers belong to the latter.

When the temperature of the atmosphere is unusually elevated and the air dry, perspiration takes place with so much readiness that it preserves the heat of the body at its normal standard. If, instead of being still, the atmosphere passes over the surface in a current, the quantity of perspiration is still further increased, and the cooling influence is felt more. But if with the same temperature the atmosphere is loaded with moisture, perspiration is prevented, and the heat of the body becomes intense.

The influence of stimulation in the promotion of perspiration is shown in the effects of exercise, the warm bath and diaphoretics. Instances of the influence of the nervous system are exhibited in the total arrest of perspiration during the hot stages of fever and in its great increase

29. Langley, J. N.: The Secretion of Sweat, J. Physiol. **56**:110 (Feb.) 1922.

under emotions of a depressing kind, such as fear and anxiety, and also in syncope. The perspiratory secretion possesses its highest amount of activity during digestion and is at its minimum immediately after the taking of food.

The easiest way to make a person sweat is to make him hyperthermic either by exercise or by hot baths. The local temperature of the sweating part also has some influence. There seems to be no doubt but that the activity of the sweat glands is more readily called forth by direct stimulation of the sweat center anterior to the tuber cinereum (Hasama [30]) when the skin is warm, for if the skin is cold the inhibition of the center must be first overcome. On the other hand, many persons sweat from the forehead before a rise in body temperature occurs.

One would anticipate that with all this outpouring of water through the skin the blood would become more concentrated. As a matter of fact, the opposite occurs. It has been shown that with the onset of sweating, there is a flow of water, salts and proteins from the tissues into the blood stream, and the blood becomes richer in plasma and poorer in corpuscles. When the sweating is profuse and prolonged, the water derivable from the muscles and the other tissues approaches the limit, and the blood concentrates. When this occurs the secretion of sweat diminishes, and the person is in danger of hyperthermia.

Experiments on the inhibition of sweating by Kuno and Ikeuchi [31] showed that when the subject was in a state of moderate sweating the application of an ice bag to any part of the skin caused a moderate decrease in sweating of the whole body surface. This decrease appeared immediately after its application but was transitory and was gradually restored, although the ice bag was still kept on the same place. If it was slightly changed to another area close by, a new decrease in sweating resulted. The inhibition of sweating on the application of cold is therefore caused in different ways. During moderate sweating it is largely due to a nervous reflex, while during extreme sweating, the effect of cooling the blood plays the most important role.

Facial ephedrosis may accompany disease of the central nervous system, as in hemiplegia, dementia paralytica, glioma, tabes dorsalis or injury of the spinal cord. It may accompany (Vaughan [32]) structural or functional pathologic changes in the cervical portion of the sympathetic nervous system, round cell infiltration of ganglions or disease of

30. Hasama, B.: Pharmakologische und physiologische Studien über die Schweisszentren, Arch. f. exper. Path. u. Pharmakol. **146**:129, 1929.

31. Kuno, Y., and Ikeuchi, K.: Sweating in Man Caused by Effect of Heat and Also Variations of Body and Skin Temperatures, J. Orient. Med. **9**:35 (Sept.) 1928.

32. Vaughan, W. T.: Unilateral Hyperhidrosis and Erosion of Teeth Following Parotid Abscess, J. A. M. A. **84**:583 (Feb. 21) 1925.

the superior mediastinum or of the cervical region. It has occasionally been reported with general debilitating diseases, such as diabetes mellitus and obscure abdominal conditions.

Adamkiewicz [33] observed that fibers controlling the secretion of sweat are present in the facial nerve. Faradic stimulation of this nerve in healthy persons causes profuse secretion of sweat in the areas supplied. Partial facial paralysis with hyperhidrosis has been associated with muscular twitching of the affected side, suggesting a pathologic irritation of the secreting fibers in the seventh nerve. On the other hand, cases have been reported in which excessive sweating has occurred in the presence of complete paralysis of the nerve. Localized sweating accompanying disease of the parotid gland is fairly common. It is usually associated with functional impairment of that organ. One usually obtains a history of abscess or injury. Occasionally the condition follows obstruction of ducts by salivary calculi.

Age appears to exert no gross effect on the activity of the sudoriparous response to dry heat; persons over 60 years of age have been seen to react easily in a few minutes without undue rise of temperature. In persons whose skin is extremely atrophic, there is apparently some delay in response. This is probably associated with and partially due to a corresponding reduction in the activity of the thyroid gland.

Hall [34] reported 2 cases in which paroxysms of sweating occurred only on chewing highly flavored substances, such as onions and pickles. He expressed the opinion that this condition suggests some hyperexcitability of the reflex sweating center for the face, possibly within the pons.

In another case, according to Weber,[35] unilateral sweating and flushing of a portion of the face occurred especially after eating. The right malar region showed localized sweating after ingestion of rhubarb and once after stewed gooseberries.

According to Hare,[36] localized sweating occurs almost solely in persons with nervous disease, often being organic, as in dementia paralytica, and sometimes functional, as in hysteria or Raynaud's disease. It depends on perverted vasomotor influences sent to the glands and their supplying vessels in particular areas. Localized sweating of

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33. Adamkiewicz, A.: Die Secretion des Schweisses, Berlin, A. Hirschwald, 1878.

34. Hall, R.: Transient Unilateral Hyperidrosis Localis Immediately Following Eating of Highly Acid Food-Stuffs, Irish J. M. Sc. **5**:291 (Oct.) 1923.

35. Weber, F. P.: Remarks on Localized Flushing and Sweating of the Cheek on Eating, Also on Striae Patellares, M. Press **130**:261, 1905.

36. Hare, C. J.: Axillary Mamma Secreting Milk, Tr. Path. Soc. London **11**: 304, 1860.

one side of the face, neck or chest is often a most important sign of a thoracic aneurysm pressing on the cervical portion of the sympathetic nervous system.

Pusey [37] reported a case in which hyperhidrosis had been present in a woman of 21 for ten years, being limited to a small patch on the extensor surface of the right arm and hand.

Of interest is Sutton's [38] case of hyperhidrosis circumscripta in an Italian laborer, in whom the involved area was in the left subscapular region and measured 8 by 18 cm. The attacks were precipitated by fright, worry or excitement. Microscopic observations indicated that in some instances long-continued localized hyperhidrosis may result in or be accompanied by hypertrophy of the coil glands in the involved areas.

Palmer [39] reported a case of angina pectoris in which the patient awoke to find the precordium, left shoulder and left arm to the elbow drenched with sweat. Pain was lacking, but the pulse rate was accelerated. The author regarded the sweating as an example of an unusual sympathetic reflex phenomenon. Mackenzie [40] likewise noted flushing or sweating of the forehead and body during anginal attacks in addition to an increased flow of urine.

Holt [41] reported localized sweating replacing cardiac pain. The sweating was limited to the region where the pain had previously been felt and was observed repeatedly in the last year of the patient's life. No other evidence of sympathetic involvement existed.

With psoriasis it has been noticed (Pemberton [42]) that sweating fails to occur on the site of active cutaneous lesions irrespective of the presence of scales. This was repeatedly observed in every case studied. Psoriatic patches that were partly healed showed less active sweat secretion than did areas of clear skin. In several patients the sweat collected from partly healed areas was more acid than sweat from uninvolved neighboring areas.

In a case of scleroderma observed for several months, the sweat nitrogen, high at first, progressively decreased coincidentally with clinical

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37. Pusey, W. A.: Case of Acquired Circumscribed Hyperhidrosis, Arch. Dermat. & Syph. **1**:438 (April) 1920.

38. Sutton, R. L.: Hyperhidrosis Circumscripta, J. Cutan. Dis. **35**:154 (March) 1917.

39. Palmer, R. S.: Localized Sweating, a Sympathetic Reflex Phenomenon in Angina Pectoris, Am. Heart J. **5**:519 (April) 1930.

40. Mackenzie, J.: Diseases of the Heart, ed. 4, London, Oxford University Press, 1925, p. 337.

41. Holt, E.: Localized Sweating Replacing Cardiac Pain, Am. Heart J. **5**: 522 (April) 1930.

42. Pemberton, R.: A Note on the Composition of Human Sweat, Ann. Int. Med. **2**:1243 (June) 1929.

improvement. A large proportion of cases of renal vascular disease are characterized by deficiency in sweating or in the vascular reaction to heat. This abnormality is commonest in patients with severe hypertension but may also be found with other conditions, especially in the terminal stages.

The conditions which respond best to sweating are presumably those in which the renal factor is more prominent than the general vascular disorder.

Pemberton [42] called attention to the value of heat in the treatment of disease. This artificial production of sweat has been so successful as to lead many to believe that toxic materials of various kinds may be thus eliminated.

CHEMICAL COMPOSITION AND OUTPUT OF SWEAT

Adolph [43] stated that the composition of sweat from the human body varies with the region and with the duration of sweating. It does not vary with atmospheric conditions. Slight variations in its reaction and chloride concentration were found to correlate simultaneously with changes in the blood.

While the composition of the sweat is correlated slightly with that of the blood, the product elaborated by the sweat glands seems to be primarily independent of the chemical conditions within the body. The sweat glands are therefore comparable to the salivary glands and the mammary glands in their work of producing a uniform secretion.

Sweat may be either acid or alkaline, with a p_H varying from 4.8 to 8.4. Initial sweat is usually more acid than that which is secreted later, if conditions favorable for sweating are maintained. This change in reaction seems to be a compensatory measure to the systemic alkalosis which develops during a "bake," hot bath or other similar exposure of the body to external heat.

Since acid sweat contains only small amounts of carbon dioxide, the alkaline swing observed during sweating is probably the result of increased elimination in the sweat of basic substances.

Numerous experiments fail to indicate that fatty acids are concerned in the reaction of sweat; also, because of the uncertainty of quantitative measurement of such small amounts of phosphorus and the unknown dilution factor of the sweat, it is extremely hazardous at present to ascribe the acidity of sweat to the salts of phosphoric acid or to any other sweat constituent that has been investigated.

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43. Adolph, E. F.: Nature of Activities of Human Sweat Glands, Am. J. Physiol. **66**:445 (Nov.) 1923.

The normal reaction of a living cell is slightly acid. Levin and Silvers [44] stated that in patients with diseases and inflammations the reaction of the cell and tissue juices becomes more acid. Schade, Neukirch and Halpert,[45] investigators of this particular phase, found that in cases of inflammation the subcutaneous connective tissue showed a p_H of 5.6, which is decidedly acid.

Talbert [46] found that sweat due to heat has a p_H of 5.73 and work sweat a p_H of 6.22 and that the acidity of sweat from covered parts of the body is slightly higher than that from uncovered parts. This factor is important in influencing the location of many cutaneous diseases.

Levin and Silvers,[47] in an attempt to explain the localization of ringworm, agreed that delayed evaporation, from whatever cause, interferes with the normal acid reaction of the cutaneous surface. Evaporated sweat through its acidity seems to act like a protective covering for the skin against bacteria. Next to the toes and feet, the areas most frequently attacked by the ringworm fungus are the axillas and the inguinal regions, which contain apocrine glands. However, apocrine glands have not been discovered in the region of the toes or feet. The alkalinity and greater concentration of the sweat cannot, therefore, be due to anatomic differences of the sweat glands from those of the rest of the body.

The changed reaction of the sweat and its enhanced value as a medium for fungous growth must be due to the mechanical and physicochemical features of delayed evaporation and trauma.

In a later paper Levin and Silvers concluded that the ringworm fungus grows best in a dark moist alkaline medium like sweat, since it contains most of the ingredients necessary for bacterial development, nitrogenous compounds, sugar and salts.

In intertriginous spaces, and especially in the presence of maceration, the reaction of the surface of the skin was more alkaline, having a higher p_H than that found in other localities.

Brill,[48] in discussing the Wassermann reaction in sweat, concluded that it is important to establish firmly whether the sweat can be regarded

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44. Levin, O. L., and Silvers, S. H.: The Reaction of the Skin and Its Secretions in Eczema, Arch. Dermat. & Syph. **25**:825 (May) 1932.

45. Schade, H.; Neukirch, P., and Halpert, A.: Ueber lokale Acidosen des Gewebes und die Methodik ihrer intravitalen Messung, zugleich ein Beitrag zur Lehre der Entzündung, Ztschr. f. d. ges. exper. Med. **24**:11, 1921.

46. Talbert, G. A.: Hydrogen Ion Concentration of Human Sweat, Am. J. Physiol. **61**:493 (Aug.) 1922.

47. Levin, O. L., and Silvers, S. H.: The Possible Explanation for the Localization of Ringworm Infection Between the Toes, Arch. Dermat. & Syph. **26**:466 (Sept.) 1932.

48. Brill, E.: Analytische Untersuchungen des menschlichen Schweisses, Arch. f. Dermat. u. Syph. **155**:199, 1928.

as a filtrate of the blood, which apparently is not the case. The Wassermann reaction in sweat is completely independent of that of the blood, appearing positive in many persons without syphilis and negative in persons with proved syphilis. The variable results in sweat are possibly due to differences in the cholesterol content.

Talbert and Rosenberg's [49] work on the effect of sweating on gastric acidity showed that in 85 per cent of forty experiments, there was a sudden drop in the free acidity when the subject had been in the cabinet from twenty to thirty minutes. This approximates the time when sweating is the most profuse.

Usher [50] in his paper on human sweat as a culture medium for bacteria drew the following conclusions: 1. Human sweat has been shown to be an excellent culture medium for certain bacteria, including the various higher fungi. 2. Comparative studies reveal that it compares favorably as such with many laboratory media. 3. The activity of bacterial growth in sweat appears to vary directly with the sugar content. The presence of pyogenic infections of the skin does not appear to increase the bactericidal properties of sweat; sufficient evidence, however, is lacking to prove this statement.

Brann [51] recently showed that the epidermis is bactericidal to Staphylococcus pyogenes aureus and also that the inhibitory substance is easily extracted, even with distilled water. When sweating is induced there is always an admixture of epithelial debris. Because the inhibition to the growth of the staphylococcus is so slight, it is felt that whatever bactericidal properties are possessed by sweat may be derived from the epithelial debris. It must be concluded, therefore, that human sweat has not yet been shown to be bactericidal to Staphylococcus pyogenes aureus or to the cryptococcus.

Marfan [52] considered artificial feeding and rickets as the causal factors of the habitual excessive perspiration seen in infants. If the sweat is of abnormal composition and is not freely evaporated, it irritates the epidermis and can cause miliaria rubra. Itching with subsequent scratching of the vesicles favors invasion of the epidermis and sweat

49. Talbert, G. A., and Rosenberg, I.: Simultaneous Study of Constituents of Sweat, Urine and Blood, Also Gastric Acidity and Other Manifestations Resulting from Sweating, Am. J. Physiol. **84**:520 (April) 1928.

50. Usher, B.: Human Sweat as a Culture Medium for Bacteria, Arch. Dermat. & Syph. **18**:26 (Aug.) 1928.

51. Brann, G.: Untersuchungen über die Bactericidie der Haut, der Haare und des Ohrenschmalzes, Klin. Wchnschr. **43**:2059 (Oct. 21) 1928.

52. Marfan, A. B.: De la sueur et des eruptions sudorales chez le nourrisson; rapports des eruptions sudorales et des pyodermatites, Presse méd. **33**:1313 (Oct. 3) 1925.

glands by pus-forming cocci and a resultant pyoderma. Treatment of this condition often fails as long as there is a constant recurrence of the excessive sweating.

Of value is the determination of the p_H of normal sweat at the time of secretion. Past opinions have been more or less conflicting. Several investigators, notably Trümpy and Luchsinger,[53] maintained that the sweat was normally alkaline. They also contended that its acidity was due to its decomposition into fatty acids.

Others, including Kittsteiner,[54] stated the belief that the sweat as secreted is normally acid. If by chance an alkaline reaction was obtained, then his interpretation was that the urea had broken down into ammonia. Talbert [55] likewise has found small amounts of ammonia in sweat and also saw the importance of a thorough cleansing of the portions of the body from which the sweat was to be collected. In his experiments, the hydrogen ion concentration was determined by both the electrometric and calorimetric methods. The conclusion reached was that sweat is most uniformly acid, although occasionally an alkaline reaction is obtained. Uric, lactic and amino acids were usually present. Others have detected the presence of formic, acetic and organic acids. It is believed that certain of the acids present are volatile. Leake,[56] using the pentabromacetone method and the method of Salant and Wise, showed the presence of a substance in human sweat which is believed to have been citric acid. Some of the quantitative analyses were as follows: in sweat due to heat there is on the average 72.8 mg. of citric acid excreted per day, according to the method of Salant and Wise; in sweat due to physical exertion the same method showed 60.7 mg. and the pentabromacetone method 70.8 mg. It is seen that the difference in the last two figures indicates a certain percentage of error.

Talbert, Silvers and Johnson [57] in twenty-eight experiments on 11 different subjects found sweat to contain sugar in varying amounts (5.6 mg. to 40 mg.), the average being 15 mg. per hundred cubic centimeters. In another series of twenty-eight experiments on 12 different subjects the concentration of the amino acids found varied from 1.66

53. Trümpy, D., and Luchsinger, B.: Besitzt normaler menschlichen Schweiss wirklich säure Reaktion? Arch. f. d. ges. Physiol. **18**:494, 1879.

54. Kittsteiner, C.: Sekretion, Kochsalzgehalt und Reaktion des Schweisses, Arch. f. Hyg. **73**:25, 1911.

55. Talbert, G. A.: Ash of Human Sweat, Am. J. Physiol. **63**:350 (Jan.) 1927.

56. Leake, C. D.: Occurrence of Citric Acid in Sweat, Am. J. Physiol. **63**:540 (Feb.) 1923.

57. Talbert, G. A.; Silvers, S., and Johnson, W.: Simultaneous Study of Constituents of Sweat, Urine and Blood, Also Gastric Acidity and Other Manifestations Resulting from Sweating: Total Nitrogen of Sweat and Urine; Total Non-Protein Nitrogen of Blood, Am. J. Physiol. **81**:81 (June) 1927.

mg. to 4.76 mg. per hundred cubic centimeters. It was impossible to show that there was any constant relation in the concentration of the amino acids in the sweat and urine.

Dewar and Gamgee [58] as early as 1871 stated that in some cases in which cystine occurred in the urine, silver coins carried in the pockets of the clothes readily became blackened. Garrod,[59] however, was unable to demonstrate the presence of cystine in the sweat. More recently Lewis [60] in a known cystinuric patient was unable to detect the presence of cystine with the colorimeter method of Folin and Looney and with Wallaston's test (formation of cystine hydrochloride crystals).

Sweating resulting from either work or heat is acid, but sweat caused by external heat is always more acid than that caused by muscular work. Many believe (Talbert [60a]) that the acidity of sweat is due to its admixture with sebaceous material, but it has been shown that sweat from the palms is acid, although this area is devoid of sebaceous glands.

The fact that sweat due to heat shows a uniformly higher hydrogen ion concentration than sweat due to physical exertion is surprising and contrary to expectation. In muscular activity there is a large increase in the acid products of metabolism, and the output of acid in the sweat can be understood as part of the mechanism for preserving the acid-base equilibrium of the body. In sweat due to heat the secretion and evaporation of the water have heretofore been regarded as an important means of controlling the body temperature, the so-called physical regulation of the heat equilibrium of the body. The fact that this sweat is acid may be considered a demonstration that sweat in human beings under ordinary dietary conditions is normally acid and that this secretion, like that of urine, helps to maintain the acid-base equilibrium of the organism.

ENDOCRINE INFLUENCES

It is clear that sweating is dependent on the activity of the sympathetic nervous system. The endocrine glands and the sympathetic nervous system are so closely bound together that changes in one must be accompanied by changes in the other. Increase or decrease of the output of one gland will result in changes in one or more of the glands.

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58. Dewar, J., and Gamgee, A.: Researches on the Constitution and Physiological Relations of Cystine, J. Anat. & Physiol. **5**:142, 1871.

59. Garrod, A. E.: Inborn Errors of Metabolism, ed. 2, London, H. Frowde and Hodder & Stoughton, 1923, p. 103.

60. Lewis, H. B.: Occurrence of Cystine, Proc. Soc. Exper. Biol. & Med. **26**:69 (Oct.) 1928.

60a. Talbert, G. A.: Effect of Work and Heat on the Hydrogen Ion Concentration of the Sweat, Am. J. Physiol. **50**:433 (Dec.) 1919.

With regard to the thyroid gland, Chevens [24] claimed to have definite clinical evidence that increased secretion is associated with increased sudoriparous activity. This may be due to the excitatory effect of the thyroid on the chromaffin system or to the physiologic necessity for increased heat loss, hyperthyroidism being invariably accompanied by an increase in the basal metabolic rate. With myxedema the reverse effect is present: a dry, harsh skin with absence of sweating even on strenuous exercise. Increased activity of the adrenal glands is associated with increased sudoriparous activity; conditions such as fear are accompanied by an outpouring of epinephrine: the syndrome described as a "cold sweat."

In several cases of toxic goiter, it was noted that both the insensible sweat loss and the metabolism were high prior to operation but that after operation, both decreased in reasonably close proportion. It appears, therefore, that the insensible loss might be of significance in indicating the lowering of metabolism following a goiter operation.

This illustrates the value of the measurement of the insensible perspiration in making an approximation of the metabolism when for practical reasons the ordinary methods of determining the metabolism may not be employed.

Benedict and Root [18] stated that refinement in the method of measurement brought out sharply the influence of body size, ingestion of food and exercise on the insensible loss. Since these factors likewise affect metabolism, it is evident that there is a relation between the insensible loss and metabolism. Patients with disease of the thyroid with a high metabolic rate were found to have a large insensible loss, and diabetic patients whose metabolic rate was low, a small loss.

THE SEXUAL ROLE OF SWEAT

Bloch [61] in discussing sexual impulse stated:

> In the higher animals the sense of smell, in the course of phylogenetic development, has attained continually greater significance in relation to sexuality; and by the fact that according to the discovery of Zwaardemaker, there exists widely diffused throughout nature a distinct group of sexual odors, the so-called capryl odors, which have a natural biological complexion with the vita sexualis. These capryl odors, which already in plants play a sexual part, are in animals and in the human species localized in or near the genital organs (odoriferous glands of the beaver, musk-ox, etc., the secretions of the male foreskin and the vagina) or in other cases are found in the general secretions, such as the sweat.

61. Bloch, I.: Sexual Life of Our Time in Its Relation to Modern Civilization, translated from the sixth German edition by M. E. Paul, New York, Allied Book Co., 1911, pp. 15 and 24.

Commenting further on the effect of increasing baldness on sexual selection, he stated:

> In certain parts of the body, especially in the armpits and in the neighborhood of the external genital organs, the thick hairy covering has been retained. This may, perhaps, be dependent upon the fact that from the axillary and pubic hair certain erotic stimuli proceed, more especially certain odors. In fact, it is possible that the hair of those regions in which strong smelling secretions were produced have played the part of scent sprinklers, analogous to the "perfume brushes" of butterflies.

Steffens, cited by Bloch,[61] stated:

> Smell is believed by some to be the principal sense of the higher animals. It represents for them their own inner world; it envelops their existence. Upon smell, wherein sympathy and antipathy are represented, is based the whole security of the higher animal instinct; for carnal desire is comprehended in this sense. Indeed, in sexual union the subjective sensation which is developed by means of smell blends completely with the objective, and from the monistic union of the two arises the intenser libido, wherein the unfathomableness of the procreative force and the whole power of sex are absorbed.

Krafft-Ebing [62] cited a case to show that contact with a person's perspiration may be the exciting cause of passionate love. At the betrothal feast of the King of Navarre (Henry III) and Margaret of Valois, the king accidentally dried his face with a garment of Maria of Cleves, which was moist with her perspiration. Although she was the bride of the Prince of Conde, Henry conceived immediately such a passionate love for her that he could not resist it and made her, as history shows, unhappy. An analogous instance is related of Henry IV, whose passion for the beautiful Gabrielle is said to have originated at the instant when, at a ball, he wiped his brow with her handkerchief.

Observation has shown us that body odors, and especially those increased by excessive sweating, are capable of attracting insects in some instances and repelling them in others. This factor undoubtedly plays an important role in the contraction of disease in tropical countries.

CHROMIDROSIS

The occurrence of true chromidrosis has been disputed at length. Some of the colors said to be assumed by the sweat are purplish blue, yellow, black, brown and occasionally green.

Red pigmentation is generally associated with a reddish incrustation of the (usually axillary) hairs, the bacteriologic examination of which reveals the condition to be due to a zooglea (Bacillus prodigiosus), which grows in rose red colonies; in cases of this condition there is often a

62. von Krafft-Ebing, R.: Psychopathia Sexualis, translated from the twelfth German edition of F. J. Rebman, Brooklyn, N. Y., Physicians and Surgeons Book Company, 1937, p. 31.

history of direct contagion. This condition, according to Heidingsfeld,[63] cannot be properly classed with the so-called chromidroses but belongs among the vegetable parasitic diseases of the skin.

In hysterical women hemorrhage has sometimes occurred in the ducts of the sweat apparatus, with the result that the sweat was blood stained (hematidrosis). This condition has been described as a form of vicarious menstruation.

Indican has been said (Mcleod [64]) to be occasionally excreted by the sweat and to color it in cases in which it is found in the urine, as in a case of severe constipation.

Certain drugs may cause colored sweat; for example, potassium iodide has been known to color sweat pink, and copper, to give it a greenish tinge.

Fox [65] reported cyanidrosis in a woman in whose sweat amorphous dark purplish granules were excreted and caused the formation of dark patches around the orbit. A bluish tinge to a woman's eyelids is believed by some to be an indication of passion.

Givens, Andrews and McClugage [66] reported a case of red sweating in which red pigment was excreted in the sweat appearing around the left eye of a boy aged 10 years and running down on the cheek. This dye as excreted was soluble in water and dilute alkaline solutions and insoluble in dilute acids and ether. Two years intervened between the attacks, which lasted about ten days.

Comby [67] reported a case of chromidrosis (black) limited to the axilla in a girl aged 14, which was characterized by severe bromidrosis. This condition had been present a long time. The pigment resembled melanin and was similar to that in another case reported by Blanchard.[68]

Storck [69] described a case in which a young woman's axilla was stained blue and in which indole was found in the sweat. The presence

63. Heidingsfeld, M. L.: The Pathology of Chromidrosis, J. A. M. A. **39**: 1519 (Dec. 13) 1902.

64. Macleod, J. M. H.: Practical Handbook of the Pathology of the Skin, London, H. K. Lewis, 1903, p. 245.

65. Fox, T. C.: On Two Cases of Chromidrosis in Which a Blue Black Pigment Exuded from the Skin of the Circumoral Regions, Tr. Clin. Soc. London **14**:211, 1881.

66. Givens, M. H.; Andrews, V. L., and McClugage, H. B.: The Excretion of Red Pigment, Am. J. Physiol. **51**:199, 1920-21.

67. Comby, J.: Chromidrose et bromidrose chez une fille de quatorze ans, Arch. de méd. d. enf. **20**:147, 1917.

68. Blanchard, R.: Observation d'un cas de mélanhydrose, Bull. Acad. de méd., Paris **58**:412 and 527, 1907.

69. Storck, J. A.: Blue Sweat (Indicanhidrosis), New Orleans M. & S. J. **62**:116, 1909.

of indole was explained by the author by presuming that when the intestine is vulnerable and the kidneys for some reason are not able to eliminate large amounts of indole rapidly enough, it finds its way through the sudoriparous glands. On coming in contact with the air it undergoes a change to indigo blue in the presence of other substances eliminated with it. The elimination of indole and skatole in the sweat might explain the disagreeable odor of the Negro.

Kelsall [70] reported a case of a Mohammedan woman aged 50 who six months after ceasing menstruation appeared to be sweating blood from the breasts. An examination revealed numerous small black specks in the neighborhood of each nipple. With the aid of a lens, these were seen to be due to fleas burrowing into the skin, and it was evident that the bleeding must have come from these small burrows.

Red sweat generally contains blood, and it is more than likely that the biblical reference "and being in an agony, He prayed more earnestly; and sweat was as it were great drops of blood falling down to the ground," [71] may have given the occurrence a notability which its rarity scarcely justifies. Nevertheless, hematidrosis certainly exists, and it has this in common with chromidrosis: It is almost invariably associated with mental excitement and nervous disturbance.

In a case recorded by von Franque [72] hematidrosis followed violent hysterical convulsions in a woman aged 45. There was a copious generalized hyperhidrosis present, but only in the parts where she had previously complained of pain was the sweat red from the admixture with erythrocytes.

Blanchard [68] reported a case of melanidrosis in a boy aged 13 in whom the pigment was intensely black and shining. The surface of the tongue was black and so was the saliva. The coloring matter on the skin was an impalpable dust, and microscopic examination showed fine black amorphous granules without crystals, fat or organisms. After thoroughly cleansing the skin and pressing from the cutaneous glands any discharge contained in them, the area was closely observed by means of a binocular lens. Clear colorless sweat exuded from the pores, spread rapidly on the surrounding skin, then became brown as it dried and gradually deepened to black. After careful examination of the colorless sweat and the black insoluble powder, it was concluded that the melanidrosis probably resulted from an oxidase in the sweat. These conclusions are interesting, because oxidase has been found to be present in excess

70. Kelsall, R.: Case of Sweating Blood, Indian M. Gaz. **62**:565 (Oct.) 1917.

71. Luke 22:44.

72. von Franque, A.: Blutungen aus den Schweissdrüsen, Wurzburg med. Ztschr. **4**:73, 1863.

in colostrum. This seems a most significant association and supports the suspicion as to the close analogy between the secretion of milk and that of sweat.

The importance of the menstrual function and of nervous excitement has been sufficiently emphasized in the cases of chromidrosis reported, and a similar hormone is likely to be present in the last days of pregnancy and in the first days of the puerperium or in disturbances of the female genital organs apart from pregnancy.

In sweat secreted under abnormal conditions there may be present on rare occasions some constituent which on oxidation by air yields a black pigment; and in colostrum also there may be some constituent which by the action of oxidase yields a black pigment, especially since colostrum is known to contain an excessive amount of oxidase. The change in the sweat takes place on the surface, while the change in the milk occurs within the ducts. The sudoriparous and mammary glands have the same genesis, and similar physiologic principles modify and control their function.

Heidingsfeld [63] expressed the belief that examples of so-called chromidrosis (excluding red forms) are anomalies of pigmentation and not glandular secretion, because the pigmented bodies of chromidrosis differ from the chromophores of ordinary pigmentation in that their contents are not finely granular but homogeneous and amorphous and that they do not decolorize with hydrogen dioxide.

It is reasonable to believe that pigment in certain cases of chromidrosis may have its origin from some point in the cutis, but whether it develops from lymph or blood vessels or preexisting cell tissue will be equally if not more difficult to determine than the present unknown source of the dermal pigment. Its apparently free dissemination in the epidermis, tissue preeminently vascularized by lymph, could indicate that pigment is derived from the lymph spaces of the cutis.

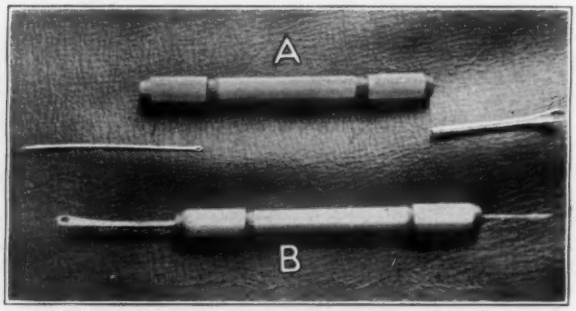
Clinical Notes

A NEW ACNE STILET

ARTHUR SCHOCH, M.D., DALLAS, TEXAS

The inability to keep a fine sharp point on the usual type of acne stilet in use prompted me to devise the instrument shown in the accompanying photograph.

The handle consists of a small vise, manufactured by a large wholesale jewelry supply house.[1] It has a chuck at each end. The needle, which is inserted in one end, is a regular Hagedorn straight surgical needle, with a fine sharp point and a cutting edge on one side. In the other end of the vise is fixed a portion of the usual acne stilet, which has been cut off and rounded to fit the chuck. The vise has approximately the same dimensions as a pocket fountain pen and is large enough to give the operator control of the instrument.



The acne stilet: *A*, dismantled; *B*, assembled.

The main advantage of this stilet is the Hagedorn needle. These needles are inexpensive and can be replaced. Resharpening is unnecessary. Sterilization is a simple matter.

The instrument lends itself equally well to extraction of comedos or to opening of the larger pustules encountered in the treatment of acne vulgaris.

1. C. & E. Marshall Company, Chicago.

SUPERINFECTION IN SYPHILIS

Report of a Possible Case

FRANK C. COMBES, M.D., NEW YORK

T.B., a man aged 44, was first seen in June 1938. Six years previously he had had an initial lesion, for which he had received some irregular treatment with an arsenical and a bismuth compound.

The Wassermann reaction of the blood was strongly positive. Physical examination gave essentially negative results. He was definitely psychopathic and homosexual in his inclinations, and treatment by several psychiatrists had failed to produce any improvement. Examination of his spinal fluid showed a strongly positive Wassermann reaction and a gold curve suggestive of early dementia paralytica. Cytologic and chemical examinations were not made.

Over a period of five months he was given tryparsamide, arsphenamine, a bismuth compound and intravenous injections of colloidal mild mercurous chloride. His general health improved, and the Wassermann reaction was reduced to 1 plus. No amount of persuasion would change his homosexual habits. He lived with a woman with whom he indulged at frequent intervals in sexual relations. She was devoted to him and would repeatedly recount her efforts to promote a normal sexual life for the patient. The Wassermann reaction of her blood was negative, and she had been advised by several physicians that the patient's condition was not infectious.

About the middle of October the patient indulged in homosexual relations. Approximately two weeks later some redness and soreness developed in his throat, together with a generalized maculopapular eruption. There was no adenopathy, pruritus or lesion of the oral mucosa other than injection of the fauces. A tentative diagnosis of an early syphilitic efflorescence was made, and Wassermann reactions of the blood were 4 plus with the cholesterolized antigen and 1 plus with the alcoholic antigen. Arsphenamine was not administered. At the time of appearance of the rash he was receiving tryparsamide. The administration of this was discontinued, and intramuscular injections of bismuth subsalicylate were resumed. The eruption faded in about a month.

On November 23 the woman with whom the patient lived presented herself with a roseola, generalized adenopathy and a positive Wassermann reaction of the blood. She said she had not had sexual contact with any one but the patient.

COMMENT

It is my purpose not to discuss the merits of this case but merely to record a possible instance of superinfection. I appreciate that there exist other possibilities and interpretations of the eruption occurring in the man. Clinically it was an early syphiloderm. The sequence of events definitely suggests a general initial immunologic response produced by the introduction of Spirochaeta pallida at a time when the immunity afforded by the original infection was greatly depressed.

Abstracts from Current Literature

EDITED BY DR. HERBERT RATTNER

THE PAINFUL SUBCUTANEOUS TUBERCLE (TUBERCULUM DOLOROSUM). ARTHUR P. STOUT, Am. J. Cancer **36**:25 (May) 1939.

Most of the small epidermal or subcutaneous nodules associated with tenderness or paroxysmal pain reported have been either glomus tumors or leiomyomas. An investigation of two thousand small superficial tumors of skin and subcutaneous tissue showed that twenty, or 1 per cent, were associated with attacks of paroxysmal pain. These twenty included the two kinds mentioned and also neurofibroma, fibroma, fibrosarcoma, keloid, dermoid cyst and benign epithelioma in a sebaceous cyst. Chondrodermatitis nodularis chronica helicis, or painful nodular growth of the ear, is mentioned by the writer but is not included in his series.

MORTALITY FROM CANCER OF THE SKIN IN RELATION TO MORTALITY FROM CANCER OF OTHER SITES: AN ANALYSIS OF OCCUPATIONAL MORTALITY STATISTICS OF ENGLAND AND WALES. KITTY K. CONRAD and A. BRADFORD HILL, Am. J. Cancer **36**:83 (May) 1939.

This study shows that as the social level declines the incidence of cancer of the skin and lip and also, though at a slower rate, that of cancer of the tongue and stomach rise steadily. The investigators refute the opinion that as the number of cancers of the skin and lip increases in any group the number of internal cancers decreases. On the contrary, their studies show that, as a general rule, when there is a high rate of cancer of the lip and skin there is also an excess of cancer of other sites.

CARCINOMA CUTIS. ERIK POPPE, Am. J. Cancer **36**:170 (June) 1939.

The author reports 198 cases of cutaneous carcinoma, in 172 of which the diagnosis was proved histologically. In 63 per cent the carcinoma was basal cell; in 28 per cent of squamous cell, and in 9 per cent of an intermediate type. In most of the cases radium therapy was used, by implantation of radium needles, by application of a radium moulage or by teleradium. Roentgen irradiation, electrocoagulation or surgical intervention was resorted to in some cases, and in 30 cases the treatment was a combination of irradiation and electrocoagulation or operation. Seventy per cent of the patients remained well for more than three years, and, excluding those who died from intercurrent disease, 81 per cent of these recovered. Radium treatment produced the most permanent and the best cosmetic results and was considered preferable to surgical procedure.

MELANOMA OF THE URETHRA. H. E. SHIH, Am. J. Cancer **36**:243 (June) 1939.

The author found only 9 reports of this rare melanoma in the literature and therefore submits the report of an additional case. The lesion was situated at the urethral orifice in a Chinese male student. The lesion is usually bluish black or purplish, firm and indurated, with a rolled, well indurated border, a cartilaginous infiltrate and a moist, granular surface. It may therefore be mistaken for a chancre in the absence of a satisfactory history. In this case the diagnosis was confirmed histologically, and the patient was still free from recurrence two years after radical surgical removal of the growth and irradiation of the regional lymph nodes.

THE BLUE NEVUS (JADASSOHN-TIECHE): ITS DISTINCTION FROM ORDINARY MOLES AND MALIGNANT MELANOMAS. HAMILTON MONTGOMERY and JAMES KAHLER, Am. J. Cancer **36**:527 (Aug.) 1939.

The blue nevus is a sharply circumscribed, round or oval, firm, slightly elevated papule or nodule, varying from 2 to 15 mm. in diameter and most frequently observed on the face, forearm or hand. It is not uncommon, usually appears shortly after birth, seldom increases in size and is deep blue, blue-black or blue-gray. It is a benign lesion but cannot always be differentiated from a malignant melanoma without histologic study. It is composed of spindle-shaped melanoblasts grouped in irregular masses in the lower two thirds of the cutis and separated from the overlying epidermis by normal cutis. The nevus cells, evidently of mesodermal origin, are elongated spindle-shaped cells with long bipolar and dendritic processes, and their pigment is melanin. They are readily differentiated from the ordinary oval nevus cell.

FOERSTER, Milwaukee.

LEUKOPLAKIA AND ALLIED MOUTH CONDITIONS. D. QUICK, Canad. M. A. J. **40**:234 (March) 1939.

Quick raises the question as to the cause of precancerous lesions in the mouth. Although they have usually been considered manifestations of local irritation, evidence has accumulated that there may be some relation to toxicity, to metabolic deficiencies or even to hormonal imbalance. The local factors, such as sharp teeth, poor hygiene, ill fitting dentures, local sepsis or the production of electric current by dissimilar metals in dentures, may be considered inciting. Syphilitic leukoplakia as well as nonsyphilitic leukoplakia and other changes in the oral mucous membrane may be directly caused by a liver deficiency. Quick regards as potentially precancerous any gross localized tissue abnormality, erosion or ulceration which has resisted ordinary simple measures of treatment for over a month. He stresses the value of early biopsy. While leukoplakia is recognized by all as a potential menace, Quick states that the chronic ulcer from local trauma is apt to be far more dangerous.

CAUTION IN ARSPHENAMINE THERAPY (REPORT OF A CASE OF PURPURA HAEMORRHAGICA FOLLOWING TREATMENT WITH NEO-ARSPHENAMINE). G. B. SEXTON, Canad. M. A. J. **40**:378 (April) 1939.

To a patient with early syphilis, Sexton administered a series of ten injections of neoarsphenamine followed by a series of twelve injections of a bismuth preparation, with no untoward symptoms. At the time of the third injection of neoarsphenamine in the second course, Sexton noticed one small petechial hemorrhage. The dose of neoarsphenamine was reduced to half, but a gastrointestinal upset occurred at the time of the injection. Several hours later there was partial paralysis and bleeding from the mucous membranes, with huge extravasations of blood under the skin at several sites. The platelet count was normal. Ten months later a mild purpuric reaction developed from a small dose of bismarsen. There was no reaction to a later test dose of mapharsen.

HEAT SENSITIVITY. H. BAKER, Canad. M. A. J. **40**:385 (April) 1939.

Baker reports the case of a woman, aged 49, who was subject to a pruritic eczematous eruption on the exposed parts of her skin, which had recurred each summer for the previous six years. The eruption was considered to be due to the sun's rays together with a lack of estrogenic substance. When the patient was given estrogenic substance, freedom from attacks was obtained.

OVER-TREATMENT IN SYPHILIS. F. E. CORMIA, Canad. M. A. J. **40**:445 (May) 1939.

Cormia describes a syndrome due to prolonged therapy with arsphenamine and with bismuth preparations and gives the detailed history of 3 patients who were observed suffering from this condition. The symptoms were referable to the nervous system (nervous irritability, insomnia and headaches), to the gastrointestinal tract (anorexia, chronic dyspepsia and constipation) and to the respiratory system (chronic cough) and also included general weakness, malaise and loss of weight. Cormia states that the current medical trend is underexamination and overtreatment of the patient with syphilis.

THE ROLE OF DIETETICS IN DERMATOLOGY, INCLUDING A DISCUSSION OF THE CUTANEOUS MANIFESTATIONS OF FOOD ALLERGY. G. S. WILLIAMSON, Canad. M. A. J. **40**:470 (May) 1939.

Williamson points out the importance of diet in therapy of the skin from the standpoints of deficiency of necessary components, particularly of the vitamins, and the necessity for restriction of certain fractions when the cutaneous disease denotes a metabolic disturbance, as in xanthoma diabeticorum. Certain foods may be harmful or of etiologic significance in diseases like rosacea, perhaps because of their stimulating influence. It is in the field of allergy in relation to the skin, however, that selection of the correct diet is of great importance. In this connection Williamson cites urticaria, angioneurotic edema, purpura, acne vulgaris and atopic dermatitis as diseases often caused by hypersensitiveness to specific foods.

A CASE OF PELLAGRA IN BRITISH COLUMBIA. F. N. ROBERTSON and D. E. H. CLEVELAND, Canad. M. A. J. **40**:584 (June) 1939.

Robertson and Cleveland report the case of a physician, aged 60, who presented the syndrome of pellagra with neurologic, gastrointestinal and cutaneous manifestations. The course was rapid, and his death occurred sixty hours after his second admission to the hospital. There was a rapid postmortem depigmentation of the skin on the backs of the hands, which had previously been deep grayish brown. Since this color is probably due to a pigmented corneous layer, scrubbing with soap and water would remove most of it. The authors did not believe it necessary to invoke an internal chemical change to account for the disappearance of the color suddenly after death.

LAWS REQUIRING PREMARITAL AND PREGNANCY TESTS FOR SYPHILIS: REASONS FOR AND AGAINST. J. A. KOLMER, J. A. M. A. **112**:2385 (June 10) 1939.

Kolmer discusses a number of important problems connected with legislation enacted by several states requiring serologic tests for syphilis before marriage and in pregnant women. He favors such legislation but advocates a medical examination as requisite to determine infectiousness. Serologic tests detect latent infection in the absence of a history or knowledge of the presence of the disease by the patient. Kolmer warns against not interpreting false positive reactions and against relying on any laboratory test as an absolute diagnostic principle.

PHOTOSENSITIZING AGENTS: A BRIEF REVIEW OF THE LITERATURE. I. L. SANDLER, J. A. M. A. **112**:2411 (June 10) 1939.

A woman, aged 30, is reported on in whom pigmentation developed on three different occasions after contact with lipstick, eau de cologne and a red rayon bathing suit, respectively. With all three different substances exposure to sunlight was considered a necessary part of the sensitization. In a second case, widespread pigmentation followed the application of eau de cologne and subsequent exposure to the sun. Test applications were made using two brands of eau de cologne (one

brand of which was the apparent cause of the pigmentation) and oil of bergamot, followed by irradiation with artificial sunlight. Pigmentation occurred at the site of application of the oil of bergamot and of the eau de cologne which had not previously been used.

THE RELIEF OF ARTHRITIC SYMPTOMS FOLLOWING URTICARIA. W. B. RAWLS, J. A. M. A. **112**:2509 (June 17) 1939.

Rawls administered cinchophen to 9 patients with rheumatoid or mixed arthritis until urticaria developed. In 5 patients partial or complete relief was obtained for from three weeks to five months, the average being nine weeks. There was no visible jaundice in any case. Rawls concluded that in cinchophen toxicity the factor which produces the urticaria may not be the same as that which produces relief of the arthritic symptoms.

PELLAGRA OF KENTUCKY MOUNTAIN FOLK: AMBULATORY TREATMENT WITH NICOTINIC ACID. J. H. KOOSER and M. A. BLANKENHORN, J. A. M. A. **112**: 2581 (June 24) 1939.

Kooser and Blankenhorn confirm the reports of others as to the efficacy of nicotinic acid in the treatment of pellagra. Of 41 pellagrins so treated and without a change of diet, 28 were completely cured and remained well, and 12 were partially improved. The usual dose was from 150 to 200 mg. a day for two or three weeks, followed by a decreased amount until the patient was free of symptoms. Dermal and mucosal lesions responded to treatment in a dramatic manner, while subjective symptoms, such as pruritus, hot mouth and burning stomach, often persisted.

HERPES ZOSTER AND VITAMIN B_1. H. RATTNER and H. C. ROLL, J. A. M. A. **112**:2585 (June 24) 1939.

Vitamin B_1 has been shown to possess antineuritic properties and has been used successfully for the relief of pain and in the treatment of various inflammatory and degenerative diseases of the central nervous system. Rattner and Roll postulated that the cause of herpes zoster and that of polyneuritis are similar. Since polyneuritis was favorably affected by vitamin B_1, a similar result might be expected in zoster. The authors treated 16 patients with herpes zoster, giving an average total dose of 5,620 international units of vitamin B_1, administered subcutaneously. No untoward reactions developed. The treatment gave indifferent results and was no more beneficial than the usual less expensive methods. In 2 patients with postzoster neuralgia no relief was obtained.

METHODS FOR CONTROLLING SCHISTOSOME DERMATITIS. S. BRACKETT, J. A. M. A. **113**:117 (July 8) 1939.

While instances of schistosome dermatitis have been reported from many parts of the United States, it is most prevalent in the lake regions of the North Central states, including Minnesota, Wisconsin and Michigan, and in the adjoining Canadian province of Manitoba. Four species of Schistosoma cercariae in Wisconsin have been shown capable of producing dermatitis. Part of the life cycle of all four species is carried out in one of several species of snails. Brackett observed that schistosome dermatitis could be controlled by killing the snails when they were in a small body of water or were concentrated in beds. He recommended copper carbonate for this purpose as a superior chemical to copper sulfate, safer and easier to use and effective in one application. It does not kill fish, whereas copper sulfate does. In large lakes where the snails are widely distributed this method is not practicable. Brackett suggested that in large lakes daily spraying, preferably at 8 or 9 a. m., of the surface of the water with a solution of formaldehyde will kill the cercariae, since they emerge at daybreak and become concentrated on or near

the surface of the water. Brackett demonstrated that Schistosoma cercariae penetrate human skin largely, if not entirely, when the water is evaporating. He observed that a bather could prevent this penetration by vigorous wiping immediately after coming from the infested water. LEWIS, New York.

SENSITIZATION OF SKIN INDUCED BY TURBINAL POLLEN APPLICATION, RESULTING IN FACIAL ACNE. I. S. KAHN, J. Allergy **10**:235 (March) 1939.

The author reports a case in which unilateral facial acne followed the positive reaction to a diagnostic intranasal application of pollen on the same side and reappeared at the sensitized site on four separate occasions in the course of specific treatment. The condition accompanied reactions at the site of local treatment and those in the nose and chest from overtreatment with the same pollen and at no other time previous, during or subsequent to the period of observation. The specific pollen was the common amaranth (Amaranthus palmeri).

UNUSUAL REACTIONS TO SLOW EPINEPHRINE. JACK COHN, J. Allergy **10**:459 (July) 1939.

In general, epinephrine base in peanut oil has distinct advantages over an aqueous solution of epinephrine hydrochloride in a dilution of 1:1,000. The author warns, however, that the use of the oily suspension of epinephrine may produce unpleasant, if not potentially serious, complications. In 3 patients with asthma and in 1 with giant urticaria epinephrine in oil in doses of 1 cc. produced the following unusual symptoms: nausea, vomiting, chills, vesicular urticaria, cyanosis, increased dyspnea and swelling and edema of the forearm. MENDELSOHN, New York.

DERMATOLOGICAL ASPECT OF THE AFFECTIONS OF THE EYE. B. B. DOWLING, Brit. M. J. **2**:794 (Oct. 23) 1937.

The following are the important causes of rosacea, in order of their relative importance:

1. The first cause is vasomotor instability, with a constitutional background, which is often inherited. This is the type of rosacea in which ocular complications are the most common. In regard to the question of ocular complications, a theory is proposed that excessive vascularization of the conjunctiva of the lower eyelids, which is part of the picture of the general increased vascularization of the skin, may have some direct relation to the more serious complications that involve the cornea in some of the cases. 2. Another cause is vasomotor instability accompanied by gastrointestinal disturbances and associated with faulty hygiene, excessive tea drinking, alcohol and lack of general recreation. 3. These factors of faulty hygiene and gastrointestinal disturbances without the vasomotor instability make up the third cause. 4. It is also caused by vasomotor changes associated with the menopause.

This article also briefly but succinctly discusses blepharitis and divides this condition into the seborrheic type and the type which is described as sycosis of the eyelashes. In men this condition is frequently associated with sycosis barbae and occasionally with chronic staphylococcic infection of the vestibule of the nose.

AUTOPHYTIC DERMATITIS. HENRY MACCORMAC, Brit. M. J. **2**:1153 (Dec. 11) 1937.

Self-inflicted inflammation of the skin may produce reactions varying from simple erythema to ulceration and deep necrosis with subsequent scar formation. The characteristic of the artificially produced lesions lies in their lack of accurate reproduction of genuine dermatoses. The cases in which artificial inflammation is superimposed on a dermatitis already present, especially in industrial dermatitis, are more diffiuclt to evaluate, because chemical agents are not always necessary to

produce an exacerbation. Sometimes merely vigorous friction is sufficient. Simple occlusive dressings in such instances are sometimes contraindicated, and immobilization can be produced by fixing the area in a wire cage. There are four principal groups of autophytic dermatoses: 1. The purposeless hysterical eruptions are almost entirely confined to young unmarried women in whom are frequently observed insensitive conjunctivas or palates and "glove and stocking" areas of anesthesia. In this group also come the hysterical and purposeful eruptions usually observed in women of mature age who have become difficult and unreasonable and are attempting to evade unwelcome duties or to gather pity with simulated illness. 2. In the malingering group there are no essential differences in the forms of eruptions except that there is a better copy of a genuine disease and there is a motive of financial gain or the avoidance of a disagreeable duty. 3. The third group is the mischief group, consisting almost exclusively of children who enjoy mystifying their elders, for instance, by pulling out hair in patches to mimic alopecia areata. 4. The last group includes the fantom dermatoses. The patient complains of an eruption or a lesion of which little or nothing is visible to the observer. These might almost come under the heading of hallucinations. The author stresses the fact that nothing is gained by confronting the patient with the deception, because the result is always an indignant denial; and even when the nature of the condition is established beyond reasonable doubt, the diagnosis is still denied by the patient and only grudgingly accepted by his friends. The exposure is never popular with any one. These conditions except in malingering patients are not diseases of the skin but rather reflections on the skin of a disordered condition of the mind.

APPEL, Boston.

MICRONODULAR TUBERCULIDS. L. M. PAUTRIER and F. WORINGER, Bull. Soc. franç. de dermat. et syph. **46**:413 (April) 1939.

The authors reserve the term papulonecrotic tuberculid for lesions which are truly necrotic or ulcerated, heal with a white scar surrounded by a pigmented zone and correspond in particular to folliclis of the extremities. The term micronodular tuberculid seems to be particularly convenient for lesions seen most frequently on the face and described under various names, such as lupus follicularis disseminatus, acinitis and (improperly) miliary lupoid. The characteristics of this type are an explosive appearance, a perifollicular dermic localization and a tuberculoid structure, with tendency to necrosis, suppuration, caseation or disintegration of the tissues. The micronodular tuberculids are sharply defined on the one hand from lupus tuberculosis and on the other hand from sarcoid. LAYMON, Minneapolis.

MULTIPLE TUBERCULOUS GUMMAS IN THE COURSE OF A PRIMARY INFECTION. E. LORTAT-JACOB and LEBENTAL, Bull. Soc. franç. de dermat. et syph. **46**:660 (April) 1939.

The patient was a girl aged 16 months with an eruption on the legs and forearms of three months' duration. The lesions were indolent firm nodules which were sharply circumscribed and not fixed to the underlying tissues. The tuberculin reaction was positive, and roentgenograms of the lungs disclosed a disseminated nodular infiltration. Inoculation of animals with material from the nodules gave positive results. The general condition was good, and there was no fever.

The authors stressed the rarity of this type of cutaneous tuberculosis as a manifestation of the primary type of tuberculosis and remarked concerning the torpid apyretic character of the general infection.

Society Transactions

DETROIT DERMATOLOGICAL SOCIETY

ARTHUR R. WOODBURNE, M.D., *President*

RUTH HERRICK, M.D., *Secretary*

FRANK R. MENAGH, M.D., *Recorder*

Oct. 17, 1939

All the presentations except that of the case of squamous carcinoma, which was made by Dr. Clyde K. Hasley, were made by Drs. Arthur E. Schiller, Clyde K. Hasley and Ray S. Dixon.

Sycosis Vulgaris.

M. A., a white man aged 34, had his jaw broken on Dec. 9, 1938, at which time the skin was broken to the right of the midline. A tooth was discharged through this break in the skin, and, though the jaw healed, a draining sinus developed. While he was still wearing a bandage, the follicular infection now present appeared. Although the sinus healed in July 1939, the eruption has persisted in spite of treatment. On one occasion the patient did not shave for one month. Since September 1939 he has been treated with hot wet packs of 1 per cent cupric sulfate continuously and with cupric sulfate iontophoresis twice weekly. This has resulted in improvement, but the hair follicles continue to show infection around the old sinus.

DISCUSSION

DR. ARTHUR P. R. JAMES, Toledo, Ohio: I accept the diagnosis of sycosis vulgaris. I believe that this man has a staphylococcic infection of the follicles. The point of particular interest in this case is the question of treatment. I, like most physicians, feel that chlorhydroxyquinoline (quinolor) ointment is the method of choice for local treatment. I have had good results with hypodermic injections of Swift's ambatoxoid, giving it in gradually increasing doses up to about 0.5 cc.

DR. CHESTER A. DOTY: About a year and a half ago this patient was treated for syphilis at the clinic of the Highland Park General Hospital. At that time he had a positive Wassermann reaction of the blood, and the spinal fluid showed a colloidal curve of 554 in the first three tubes. He also had a draining frontal sinus and this staphylococcic infection on the upper lip. He is a heavy drinker, and his jaw was broken while he was under the influence of alcohol. I agree with the diagnosis. The condition is definitely a staphylococcic infection.

DR. ARTHUR E. SCHILLER: We have had a surprising amount of difficulty in clearing up sycosis vulgaris. Chlorhydroxyquinoline ointment would work for a week or two; then a dermatitis would develop, and the patient would be right back where he was before. Recently we have been using cupric sulfate iontophoresis, using 1 per cent of cupric sulfate, together with wet packs of aluminum acetate. We have about come to the conclusion that the majority of patients with sycosis vulgaris are overtreated and that while chlorhydroxyquinoline is an excellent preparation for some of them, it should be used only for the quiescent conditions rather than for those that are active.

Squamous Carcinoma (Bowen's Disease?). Presented by DR. CLYDE K. HASLEY.

The patient is an elderly white man, who had been treated for two years for psoriasis before he presented himself on April 28, 1939, complaining of a mass on the left thigh. The tumor was about 2½ inches (6.3 cm.) in diameter and was elevated about 1 inch (2.5 cm.). The surface was ulcerated, and there was a palpable gland in the left inguinal region.

On the neck were two superficial lesions that were thought to be epitheliomas of the Bowen type. The lesion on the thigh had started as a superficial lesion similar to those on the neck.

Histologic examination of the lesion on the thigh showed a squamous carcinoma.

The patient is shown to demonstrate the results of filtered and unfiltered roentgen ray therapy. The patient received the following dosage:

Unfiltered roentgen rays (April 28 to May 15)	2,865 r
Filtered roentgen rays, 0.25 mm. copper and 1 mm. aluminum	2,913 r
Total	5,778 r

DISCUSSION

DR. CLYDE K. HASLEY: This case was presented for two reasons: first, to convince some of the members that it is perfectly safe to use unfiltered roentgen rays in large quantities and, secondly, to discuss the question of dosage.

This man received 1,710 roentgens of unfiltered roentgen rays in one treatment; four days later he received 630 r of unfiltered rays, and two days later he was again treated, receiving 525 r. After that, filtered roentgen rays were used, the filter used being 0.25 mm. copper and 1 mm. aluminum, with 120 kilovolts. He received 578 r of filtered radiation in addition to the 2,865 r of unfiltered rays.

A word of caution should be added. Clinically this lesion looked like mycosis fungoides. The roentgen ray dosage was measured in air. With the back-scatter in a lesion of this type, approximately 35 per cent more radiation is received than is measured in the air. In other words, this man received approximately 6,000 r plus one-third this amount.

What his skin will look like in two or three years remains to be seen. The only thing I can say is that I have used this treatment before, and it did not cause telangiectasia. Occasionally changes are seen in the normal skin but not in the area occupied by the lesion.

Another thing I should like to discuss is the architecture of the histologic section. The patient presented other lesions on his neck that I diagnosed as Bowen's epithelioma, and I believe that this lesion was a degenerated Bowen epithelioma. Dr. Owen thought that the section showed the architecture of Bowen's disease. Because the base of the lesion was shown in one area to extend below the epithelium, a diagnosis of squamous cell epithelioma was made. The patient was told that he had psoriasis, but no psoriasis is now evident. He has multiple cancer, as there is also a squamous cell carcinoma of the throat. This has been treated with roentgen rays.

DR. CHESTER A. DOTY: You say he has carcinoma of the throat now?

DR. CLYDE K. HASLEY: Yes, he has a malignant growth in the larynx.

Lupus Erythematosus.

The patient, a Polish laborer aged 45, was seen because of an eruption of four years' duration, which first appeared on the chest during the summer and then spread to the face. It improves during the winter but has never completely disappeared. During July 1939, after exposure to sunlight, all the old lesions flared, and new ones appeared.

On examination, the patient presented dully erythematous, infiltrated, scaly patches on the ears, malar eminences, entire nose, upper lips and chest. There are also small isolated scaly patches on the back of the neck and the lower portion of the scalp. The lesions are not pruritic.

With the exception of carious teeth, the physical examination gave negative results.

The Wassermann reaction was negative. The urine and blood were normal.

The patient has had sulfanilamide therapy, without success, and is now receiving injections of bismuth subsalicylate.

DISCUSSION

Dr. George Van Rhee: I have a case of a young girl who has discoid disseminated lupus erythematosus with acute flare-ups, which is not the exanthematous type, whom I have been able to keep going for almost two years with rather frequent blood transfusions. She has done well, being practically free from lesions for nearly nine months, although I do not know how long this freedom will continue. She did not respond to any other therapy, and a gold compound even caused the eruption to flare up.

Dr. Frank R. Menagh: In this connection, I have seen several patients who were sensitive to gold sodium thiosulfate but who were able to tolerate sodium succinimide aurate, as advocated by Dr. Becker. The clinical results were as good as they were with the gold sodium thiosulfate.

Dr. Arthur E. Schiller: The thing I should like to comment on is the increase in the number of cases of lupus erythematosus observed this past summer. In each case the exposure to sunlight was given as a dominant factor in the production of the condition. During the hot weather, when there was a great deal of sunlight, it seemed impossible to get good results from treatment, regardless of the method used.

We had 1 patient, who should have been here today, whose skin with the same treatment, which was a failure during the time he was exposed to sunlight, cleared up brilliantly as soon as the colder weather started and he began working indoors.

I think that the increase in incidence of lupus erythematosus in Detroit and in Michigan is worthy of mention.

Dr. George H. Belote, Ann Arbor, Mich.: I presume Dr. Schiller was speaking of the discoid type of lupus erythematosus. I should also like to remark that I have observed a considerable increase in the number of cases of the exanthematous type in the past year. I have 6 or 8 patients with the condition, and they have been treated with every method that I know and every method that any one else has proposed, including medication with large doses of sulfanilamide and all its derivatives, and the results obtained have all been the same. I am thoroughly discouraged about the treatment of lupus erythematosus of the exanthematous type. In cases of the discoid type, recovery is observed at times, with various types of treatment. Like Dr. Van Rhee, I am a little partial to the gold compounds for the discoid type, but even with gold compounds the conditions sometimes disseminate. It has been my feeling that sulfanilamide has done absolutely nothing for the acute disseminated exanthematous type. I have thought that I have seen some improvement in the discoid type following the use of sulfanilamide, but I have also used its derivatives, with little result.

Dr. George Van Rhee: I agree with the diagnosis but not with the treatment. I do not believe that sulfanilamide will accomplish much in cases of lupus erythematosus of the discoid type, and I do not believe that bismuth will accomplish much. I have seen little improvement resulting from injections of bismuth preparations over a long period. I think there is only one form of treatment for that man, and that is the administration of gold sodium thiosulfate in good-sized doses, with a gradual increase up to 100 mg. a dose.

Scleroderma.

A girl aged 6 years, born in the United States, first came to the Grace Hospital clinic about three years ago, presenting an infiltrated smooth yellowish glistening patch above the inner surface of the left knee. This patch rapidly extended

upward until the skin of the entire thigh, the abdomen and the chest up to the nipple were involved. Within six months the entire area took on a waxy, glossy appearance, which was more severe around the knee and the lower part of the thigh. The lesions then appeared around the outer part of the left thigh and hip, and various small patches of morphea appeared on the back, shoulders and chest. The latter lasted about two or three months and disappeared, with atrophic areas remaining.

On Aug. 9, 1939, the measurement of the legs showed: left, 23 inches (58.5 cm.), and right, 23⅜ inches (59.5 cm.) (measuring from the spina iliaca posterior superior to the inner malleolus). A measurement on September 30 showed a shortening of ¾ inch (1.9 cm.).

A general physical examination of the patient gave negative results. The child is well developed but is subject to winter colds.

The patient's mother and father are both living and well. The girl has had chickenpox and measles.

The blood cholesterol content was 197.4 mg. per hundred cubic centimeters. The results of other laboratory tests were within the range of normal.

The patient was treated with thyroid and ultraviolet irradiation and responded in a measure. The skin became softer, and the infiltrations were replaced with scar tissue. For the last eight weeks she has been receiving iontophoresis twice weekly, with mecholyl (acetylbetamethylcholine hydrochloride), and is still taking 1 Gm. of thyroid per day.

Scleroderma.

The patient, a white woman aged 40, born in Poland, began to have a swelling of the right ankle about four years ago. The part would swell during the day and go down during the night. The swelling caused itching but no pain. She also had a swelling of the right ankle and leg nine years ago, after the birth of a child. The swelling has continued at times for the past four years. Shortly before Christmas in 1938 the skin of the right ankle began to be rough, and the foot and ankle were swollen at the time. The itching became worse, the foot feeling cold most of the time. Sharp pains were then felt in the area, similar to those caused by needles pricking the skin. The patient first came to the dermatologic clinic about January 1939. The symptoms were more pronounced then. She was given ointment to use on her foot. Later ultraviolet irradiation was given. The condition continued, and the patient was hospitalized in May for thirteen days.

No active pathologic changes were noted in the chest, and no gross pathologic changes in the pelvis. The woman is now going through the menopause.

About two years ago she had pneumonia. Since that time she has had a cough at times. A roentgenogram of the lungs showed bilateral apical caps, with hilar lymphadenopathy and a few calcifications, but no active tuberculous lesions were seen. Studies of the gallbladder and the gastrointestinal tract have revealed no abnormalities.

The Kahn and Kline reactions were both negative, while the results of blood tests were normal. The urine was normal. The sputum showed no acid-fast organisms.

The patient has been receiving mecholyl iontophoresis and ultraviolet treatment since leaving the hospital. No swelling of the foot is present at this time. The cutaneous condition is much improved, and there is no swelling of the right ankle. The area around the right ankle is indurated and firm, most markedly on the lateral surface. The skin is dry and rough. The right foot is colder than the left. The superficial veins are hard and indurated.

Generalized Scleroderma.

The patient, a married white woman aged 65, born in the United States, presents lesions covering the entire body. The skin is dry, glistening, shiny and tightly stretched where it has not atrophied. Numerous pigmented areas and dead

white linear scars are present on the face and neck. There is ectropion of both lids. The corners of the mouth are pulled tight. The condition is not as extensive from below the breasts to the hips, but the legs, thighs, ankles and feet show involvement. The hands are deformed, the skin being tightly stretched over the hands, arms and face. There have recently been appearing large bullae filled with clear serum, which become purulent in a few days and then break down, leaving shallow ulcers, which heal slowly.

The disease started thirteen years ago and in spite of treatment has been progressive.

DISCUSSION OF CASES OF SCLERODERMA

DR. HARTHER L. KEIM: I should like to suggest that the second case of scleroderma be considered separately; I believe that the condition is an example of circulatory deficiency, following a definite history of freezing, which has resulted in some cutaneous changes which give the picture of scleroderma. The other 2 cases I think are examples of scleroderma.

DR. HOWARD J. PARKHURST, Toledo, Ohio: In the second case listed as one of scleroderma acrodermatitis would have to be considered. The skin is transparent, with a cigaret paper appearance, showing the blood vessels, clinically the picture of acrodermatitis. It is possible that the terminal, atrophic stage of scleroderma might be present, but I favor acrodermatitis chronica atrophicans.

DR. ARTHUR R. WOODBURNE, Grand Rapids, Mich.: I think I should have to back Dr. Keim's opinion, in preference to that of Dr. Parkhurst. It does not appear that the typical atrophy of acrodermatitis is here. The lesions show a dusky diffuse erythema, which I class as a vascular disturbance of the pernio type.

DR. FRANK STILES, Lansing, Mich.: I should like to raise one question. In the first case presented, were those little spots on the back and shoulders of the girl considered to be part of the scleroderma? They were small lesions, about half the size of a pea, depressed but not sclerotic.

DR. ARTHUR E. SCHILLER: Each one of those patches at one time was a definite morphea. The girl was shown to this group about three years ago, and the picture has changed considerably since that time. The point worthy of comment, both in the girl and in the elderly woman, is the crippling effect of scleroderma. The girl has a shortening of the leg, and the elderly woman has definitely crippled hands. Her ankles are so hidebound that she can walk only with difficulty, and the skin of the face is so hard that it cracks and breaks when there is the slightest amount of injury. On the skin over the hands enormous blebs develop, some of which eventuate as pustules and ulcers.

DR. UDO J. WILE, Ann Arbor, Mich.: One is always inclined to regard morphea as an unfortunate but rather inconsequential condition. Ordinarily, the lesions of morphea disappear, leaving more or less atrophy. In this case, however, there is a more serious aspect of the disease in the occurrence on one leg of a lesion of definite scleroderma, which approaches the generalized type. It is interesting to speculate as to whether this generalized sclerodermatous lesion was preceded by definite patches of morphea. I should like to ask Dr. Schiller whether the lesions on the elderly woman which are now keratotic were examined microscopically. It seemed to me that some of them might show epitheliomatous changes. Such changes have been noted in cases of scleroderma in addition to ulcerative lesions.

DR. ARTHUR E. SCHILLER: I agree with Dr. Wile. A number of lesions appeared to me to be definitely epitheliomatous. No biopsies have been performed yet.

The condition started on the ankle and then gradually ascended on the inner surface of the leg. Two weeks after that the entire outer surface of the thigh up to the axilla presented the particular brawny consistency of generalized scleroderma. A week or so after this she had multiple patches of morphea over the back, abdomen and chest, and there was one lesion under one ear.

DR. LOREN W. SHAFFER: I agree with the diagnosis of scleroderma in the elderly woman. However, there is some suggestion of poikiloderma, and this brings out the possible interrelation, at times, of these lesions. She has a peculiar retiform type of pigmentation, probably due in part to senility and in part to scleroderma. However, it suggests a radiodermatitis. The bullous lesions would also fit into this picture.

Eczema Seborrhoeicum, with Unusual Persistent Localization.

The patient, a white woman aged 18, born in the United States, first presented herself in August 1937, complaining of an eruption on the chin and around the alae of the nose.

She presents a vesicopapular, reddish, confluent eruption on the entire chin, rather sharply limited below and somewhat flaring on the sides, extending upward to the lower lip. The skin of the upper lip, the alae and the sides of the nose and the area between the eyebrows and forehead are also involved in a papular eruption with scaling. The scalp shows yellowish crusting in areas. The ears have not been involved, nor has the body. She gives no history of having used depilatories, cosmetics or other types of medication except that prescribed by a physician. She states that the prescribed medication did not make the condition worse.

DISCUSSION

DR. RUTH HERRICK, Grand Rapids, Mich.: In spite of the diagnosis, seborrhea, and in spite of the look of the patient's scalp, I think perhaps one can evoke an allergic reaction for this woman, and I should like to have patch tests made.

DR. CYRIL K. VALADE: Perhaps she also has some sebaceous changes going on. I remember patients with similar conditions whom I have treated with that in mind and whose skins cleared up under this therapy. The localization suggests fondling the chin.

DR. FRANK R. MENAGH: The temperature of the skin in the involved areas of the woman's face is much lower than that of the normal skin. This fact suggests a circulatory change such as is characteristic of rosacea.

DR. HARTHER L. KEIM: This patient has had an eruption for four years, and it is extremely difficult to make a definite diagnosis. One does not get an impression of ordinary seborrheic eczema. I cannot feel, either, that the condition is simple rosacea. On close examination, I thought on the edge of the active eruption that there were some small papules which had a brownish discoloration. These suggested to me some possible form of tuberculosis which has not yet reached the point at which a diagnosis can be made. The possibility, perhaps, of a Lewandowsky type of rosacea suggests itself, and I think that this should be kept in mind if the eruption persists.

DR. HOWARD J. PARKHURST, Toledo, Ohio: I agree with part of Dr. Keim's remarks. I was struck by the sharply outlined halo of normal skin immediately about the mouth. I saw this pigmentation later, and I wonder whether a diagnosis of *érythrose peribuccale pigmentaire* of Brocq might not be suitable.

DR. GEORGE H. BELOTE, Ann Arbor, Mich.: I do not think the question of a contact dermatitis can be ruled out. I rather agree with Dr. Herrick. I think the case will require further study.

The patient has bright nail polish on and certainly uses a nail polish remover sometimes. It is surprising how many eruptions around the eyes and nose are actually due to nail polish or to polish remover. I think the eruption would stand further investigation along that line.

DR. ARTHUR R. WOODBURNE, Grand Rapids, Mich.: If the history is accurate, one ordinarily does not expect an allergic eruption to be as constant and persistent as this one has been. Allergic eruptions appear and disappear more than the history suggests this eruption has done.

Primary Atrophy of the Optic Nerve.

J. R., a married Negro aged 39, first appeared at the clinic in June 1936, giving a history of loss of vision of five months' duration. The visual examination showed ability to count five fingers with the right eye at 17 feet (5.1 meters), and with the left eye at 5 feet (1.5 meters). The Wassermann reaction of the blood was positive.

Examination of the spinal fluid showed 140 cells and a positive reaction for globulin, and the Kahn, Eagle and Kline reactions were all positive.

On Feb. 4, 1937, the Kahn reaction was doubtful, the Eagle reaction positive and the Kline reaction doubtful.

On September 20 the patient complained of shooting pains in the legs.

On July 13, 1939, the Wassermann reaction of the blood was negative. On July 20 the spinal fluid contained 2 cells. The Kahn and Kline reactions were negative.

The patient was given insoluble bismuth and potassium iodide and after loss of vision received neoarsphenamine and tryparsamide.

Interstitial Keratitis.

R. O., a Negro aged 14, has congenital syphilis. He is the sixth of 7 children, the seventh having died at birth. The remaining 5 are girls, the fifth of whom is syphilitic. The boy gives a history of pain around the eyeball, with progressive loss of vision until March 1939, when he could no longer continue in the regular school.

He first appeared in the clinic on May 25, 1939, at which time he had bilateral opacity, with a positive Wassermann reaction of the blood.

The vision is improving slowly under medication with iodides, a bismuth compound and arsenic.

Bilateral Atrophy of the Optic Nerves.

R. T., a married Negress aged 37, noticed defective vision in October 1937, and approximately two years later, in July 1939, she was totally blind. She had a miscarriage in 1918 and another in 1934.

On June 26, 1939, the Wassermann reaction of her blood was positive, and the Kahn and Kline reactions of the spinal fluid were negative. Of 173 cells in the spinal fluid, 28 per cent were polymorphonuclear leukocytes and 72 per cent were lymphocytes.

On October 2 the results of examination of the spinal fluid were as before; the colloidal gold curve, not determined on the previous occasion, was 2221110000.

The patient has received three injections of an insoluble bismuth preparation.

As a result of chorioretinitis on the nasal side of the right eye, there is a sizable opening, through which the sclera may be plainly seen.

Chorioretinitis.

H. D., a white girl aged 12, is the fifth of 7 children, the first 4 having died and the sixth and seventh being presented with the mother today.

The girl entered the hospital on April 13, 1939, with a history of pains in the legs and left knee, swelling of the feet and loss of strength and of weight.

Physical examination disclosed anterior and posterior adenopathy, a fixed right pupil, chorioretinitis and tenderness of the shins.

The Wassermann reaction of the blood was positive.

Three of the other 4 members of the family are presented, indicating that the mother may transmit syphilis to her offspring for fifteen years. One child is 11 and the other 9 years old.

The mother is 36 and had an active palmar syphilid when admitted to the clinic in June 1939.

Beginning Atrophy of the Optic Nerves.

I. D., a white married woman aged 50, with a history of a miscarriage in January 1937 after seven months of pregnancy, is presented.

On her admission to the clinic in December 1937, the only definitely suggestive finding, aside from the positive serologic changes, was a sluggish reaction of the pupils.

On Sept. 21, 1939, the spinal fluid was found to contain 1 white blood cell; the reaction for globulin was positive, and the Kahn and Kline reactions also were positive, with a colloidal gold curve of 4421000000.

Examination of the fundi revealed slight blurring of the upper border of both disks. The visual fields showed moderately contracted perimeter in both eyes, with a color field showing a contracted sector.

DISCUSSION OF CASES OF OCULAR SYPHILIS

DR. HARTHER L. KEIM: It is rather stimulating to observe cases of active ocular syphilis. Outside of the large clinics in Detroit, the ophthalmologists are having some difficulty in unearthing cases of typical ocular syphilis, just as the dermatologists sometimes have trouble in demonstrating early syphilis. I was particularly interested in the case of the girl of 15 with the chorioretinitis, especially in the left eye, coming on as it did and with the rest of the family pretty well syphilized. The lesion on the mother's palm was supposedly syphilitic, a recent manifestation of a long-continued active infection. I think no physician likes to encounter cases of atrophy of the optic nerve in his office, and the white woman apparently presents a problem in therapy. The visual fields are contracting, and I think she has beginning atrophy of the optic nerve as well as mild retinitis. She has had all the standard therapy, and with the presence of her marked aneurysm the problem of further therapy presents itself. For one thing, I should like to get the ophthalmologists' opinion as to the possibility of using tryparsamide in this particular case. If they would agree to this type of therapy, I believe it would be less dangerous than the use of hyperpyrexia with a fever machine or the use of malaria fever. The other possibility is to give the patient potassium iodide and wait and see what happens.

DR. LOREN W. SHAFFER: I am, in my position at the clinic, extremely concerned about the question of atrophy of the optic nerve. The situation is such that it is practically impossible to hospitalize patients for fever therapy, unless they have symptoms of mental disease and can be committed to Eloise Hospital, Eloise, Mich., or some similar place. Some of these shortcomings may be remedied through the City of Detroit Receiving Hospital.

As Dr. Keim brought out, the possibility of the use of tryparsamide in these cases is one to which I have given a lot of thought. In the first place, recent ophthalmologic literature indicates that tryparsamide is not particularly contraindicated in cases of atrophy of the optic nerve. At least, according to reports, tryparsamide is giving better results than any other treatment short of fever therapy. Faced with the impossibility of giving fever therapy, what is one going to do? I should like to have courage enough, and also to have some one give me a little backing, so that I could follow these cases of beginning atrophy of the optic nerve and cases of the later stages as well, administering tryparsamide, realizing that I have nothing better to offer, and then see what the results would be. The patients could be carefully checked by an ophthalmologist. Certainly one might as well do nothing as give them the routine treatment now used at the clinic.

The patient with complete atrophy of the optic nerve had a strongly positive reaction of the spinal fluid when he started treatment in 1936, and at the present time his spinal fluid and blood are normal. In spite of that, he started with a mild atrophy of the optic nerve and went on to complete blindness. That brings up the point that so many patients who finally become completely blind seem to have a tendency toward a spontaneous remission of serologic symptoms. The question brought up in regard to spinal fluids with reactions of the dementia paralytica type is interesting. One patient at the City of Detroit Receiving Hospital has advanced syphilitis aortitis, nephritis and beginning atrophy of the optic nerve

but normal spinal fluid. My decision was to start treatment with the administration of tryparsamide and follow his course by careful clinical checks. One cannot use heavy metals, and I do not want to use the ordinary types of arsenicals because of their effect on the myocardium, but this case was an exception. I should like to feel justified in trying this treatment in a series of patients in the clinic, under the supposition that it is the best treatment that can be given them.

Dr. Frank R. Menagh: I have had some experience with artificial fever therapy in these cases, and as nearly as I can tell the results are about the same as they are with malaria fever therapy. In cases of acute syphilitic ocular disease, results are obtained more quickly than in those of longer-standing conditions. I have become alarmed about the use of tryparsamide for patients with atrophy of the optic nerve, even when the condition is of the early type. I have been impressed with the large number of patients with syphilis of the central nervous system who have beginning constriction in their visual fields. More of these patients eventually show clinically evident visual damage under tryparsamide therapy than when the arsphenamines are used. It is possible that the use of vitamin B as recommended may reduce the toxicity of the tryparsamide. I believe that the routine use of tryparsamide for these patients, however, would increase the amount of damage to the vision.

Dr. Udo J. Wile, Ann Arbor, Mich.: Dr. Shaffer's dilemma regarding giving these patients malarial therapy might possibly be met in this way. It seems to me that the possibility of saving a person from blindness might justify the taking of a greater risk than would otherwise be wise. If Dr. Shaffer has an available nursing service, there seems to be no reason why patients with atrophy of the optic nerve should not be inoculated with the malarial parasites, sent to their homes and then followed up by the visiting nurse. Such a regimen, so far as I know, has not been attempted on a large scale. Patients have occasionally been inoculated with malarial parasites and sent home in the care of their physicians. Precautions are relatively simple: plenty of fluid intake and sodium chloride to make up for the loss in sodium chlorides; the paroxysms should be stopped only if an emergency, such as sudden drop in blood pressure, occurs or if hyperpyrexia, temperature of over 106 F., occurs. It seems to me that a beginning could well be made in Dr. Shaffer's material, particularly in those cases in which a real emergency, such as a saving of eyesight, is at hand.

Dr. Loren W. Shaffer: One would have to give this treatment in the winter, when there are no mosquitoes around.

Dr. Cyril K. Valade: When I was with Dr. Harrison, we had a number of cases of atrophy of the optic nerve, and we used about the same form of argument to get the ophthalmologists to allow us to continue arsenical treatment as Dr. Wile has just used. Relative to the use of malaria fever therapy, Dr. Hyde, my former colleague, had a large number of patients whom he inoculated at Eloise Hospital with malaria parasites and allowed to go home for the chills under his supervision. I believe that he did not have one death as the results of this practice.

Dr. George H. Belote, Ann Arbor, Mich.: Hardly a week goes by that I do not inoculate a patient with malaria parasites for some outside physician and let the physician take care of the patient in the home.

Dr. Ray S. Dixon: In this list of patients that we presented today, only 1 is likely to benefit much from such treatment. When the patient came in, he had a rapidly progressing atrophy of the optic nerve of five months' duration, with interstitial keratitis. The boy did not drop out of school and report for examination or treatment until he was unable to see his book. The woman did not present herself until she was totally blind, and now she refuses to come in for any type of therapy. No one suspected the girl of having syphilis until a roentgenogram of the shin bone disclosed a gumma. You have seen how many times the mother has been delivered of syphilitic children, and no one suspected the presence of the disease. Probably the best opportunity is with the last patient of the 5, who has beginning atrophy of the optic nerve. However, a cardiac complication may interfere with results here. From the standpoint of ocular mani-

festations, this is the only case of an early stage. If we are to be of service to this group of patients, routine examinations of the blood and eyes will be necessary. This will bring the patients to the clinic and under treatment when treatment is effective.

I am anxious for Dr. Shaffer to try this method, because there are many patients needing this type of care. There is not money or space enough in the hospital to do the job. If he is successful, others may make use of the method.

DR. CHESTER A. DOTY: About ten years ago I treated 7 or 8 patients with the Swift-Ellis method. Among those there were 3 with atrophy of the optic nerve. I gave them only two or three treatments apiece, but it did check the atrophy. I am wondering if that form of treatment should not be considered in these cases.

NEW ENGLAND DERMATOLOGICAL SOCIETY

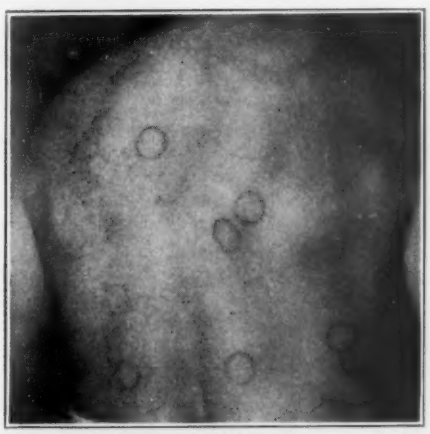
J. HARPER BLAISDELL, M.D., *President*

BERNARD APPEL, M.D., *Secretary*

Hartford, Conn., Oct. 18, 1939

Contact Dermatitis from Rubber and Alcohol. Presented by DR. C. W. O'NEILL, Hartford, Conn.

N. McC., a painter aged 62, had an occupational dermatitis in 1936, dermatitis exfoliativa of the face, hands and feet in 1937 from either kerosene or alcohol and dermatitis venenata in the same areas in 1937. For four years he has had an



Contact dermatitis from rubber and alcohol.

eczematoid dermatitis of the arms and legs. After the application to the chest of a stethoscope, which had a hard rubber bell and had been dipped in rubbing alcohol, there developed one day later, on Aug. 31, 1939, ringed lesions that were raised, with a sharply demarcated border, vesicular, papular and accompanied by severe itching. On October 16 a test with the dry stethoscope was repeated on the chest.

The patient now shows circular, crusted, erythematous, raised lesions, with about 2 inches (5 cm.) of surrounding erythema, over the manubrium. This is the

site of a twenty-four hour patch test with the dry stethoscope bell tip made of hard rubber. Patch tests with alcohol alone also gave positive results.

The ringed lesions cleared up quickly with the use of a simple soothing lotion.

DISCUSSION

DR. M. J. STRAUSS, New Haven, Conn.: How long was the stethoscope kept in contact with the skin?

DR. C. W. O'NEILL, Hartford, Conn.; The stethoscope was kept on the skin one day for the patch test. In the photographs, lesions are seen where the stethoscope was kept on momentarily while the intern examined him.

DR. M. J. STRAUSS, New Haven, Conn.: Has the stethoscope been tried on any one else?

DR. C. W. O'NEILL, Hartford, Conn.: No, the stethoscope has not been used on any one else. The patient has been a painter and has had a positive reaction to a patch test with rubbing alcohol, and he had dermatitis exfoliativa a year ago, which was due to either alcohol or kerosene or to some of the materials that he was using while painting. So I do not think the fact that the stethoscope was not used on any one else is important, because I believe he has a polysensitivity.

DR. J. H. SWARTZ, Boston: What part of the stethoscope was applied to the patient?

DR. C. W. O'NEILL, Hartford, Conn.: The tip of the bell. The composition is probably hard rubber, and there are a number of constituents in hard rubber with which he may be tested. He may be tested here with methenamine but not with the other constituents of rubber, as they are not available.

DR. J. G. DOWNING, Boston: This case presents an interesting sequence of sensitivity. The patient worked as a painter for thirty-five years, contacting many types of solvents, especially denatured alcohol. A few years ago he suffered from dermatitis exfoliativa, which was relieved; since then he has had a more or less persistent eruption on various parts of the body, especially the hands and forearms. When he entered the hospital for treatment of a pulmonary disturbance, auscultation was done with a stethoscope which had been dipped in alcohol many times. I believe that this solution was absorbed by the bell of the stethoscope to cause the positive reaction, for the man is undoubtedly sensitive to denatured alcohol. Therefore, it would be well to test him with the various constituents of the alcohol and then secure another stethoscope of the same make and test him with this, in order to determine whether he is sensitive to the constituents of the alcohol or to some rosin constituent of the compounds of which this stethoscope and bell are made.

DR. C. W. O'NEILL, Hartford, Conn.: The reaction showing today is from a patch test without any alcohol on the stethoscope. The previous pictures taken of the chest were with alcohol on the stethoscope, and the ringed lesions on his chest appeared after it had been used.

DR. J. H. BLAISDELL, Boston: This is a most interesting case, and if I had not seen the condition, I would not believe it possible. To my mind, it would be remarkable if the hard shell of the stethoscope could absorb enough of the alcohol to provoke a reaction. On the other hand, it is quite possible that the shell contains a rosin as well as a rubber compound, in which case the alcohol acts as a solvent of the rosin. The striking photographs make a worth while case to report if the causal agent can be proved.

A Case for Diagnosis (Mycosis Fungoides?). Presented by DR. E. M. STANDISH, Hartford, Conn.

A. B., a man aged 55, has had a dermatosis on his legs, lumbar region, scapular region, anterior and mesial surfaces of the thighs, arms and sides of the chest for five or six years. It began with small lesions, some of which he believes have disappeared. There was no itching, as far as he remembered, until lesions were

present. The degree of "lumping" varied from time to time. The pruritus has varied, sometimes being severe. He has felt well, although he is nervous. On September 23, while still under treatment for the original condition, typical pityriasis rosea appeared on the trunk. This has since undergone involution after one treatment with ultraviolet irradiation.

The blood smear showed 3.5 per cent eosiniphils and 3 per cent myeloblasts; otherwise it was normal.

He now shows on his arms, back, thighs and legs lichenified, extremely rough, papular to nodular lesions, most of which are grouped and coalescent. Most of the areas are excoriated. In some there is a verrucous tumor formation. Adenitis has been present only at the elbows.

After seven biweekly roentgen ray treatments, consisting of ¼ to ⅜ skin unit (75 and 112 r) to each area, the lesions improved considerably.

DISCUSSION

DR. MILDRED L. RYAN, Brockton, Mass.: I think that this patient has hypertrophic lichen planus. He shows typical small glistening papules on his shoulder.

DR. B. APPEL, Boston: I should like to suggest the diagnosis of chronic lichenoid dermatitis, which is closely related to the type which Dr. Mildred Ryan has suggested, principally because several lesions show the fairly characteristic follicular papular distribution, particularly the area over the left scapula.

DR. M. J. STRAUSS, New Haven, Conn.: The varying opinions in this case cause me to wonder if the fact has not been lost sight of that this patient has had a good deal of roentgen ray treatment and that the lesions seen today are undergoing involution, modified by roentgen ray treatment. The patient tells me that one of the lesions was at one time a large tumor. Certainly I should not make a diagnosis of mycosis fungoides on clinical grounds today.

DR. E. M. STANDISH, Hartford, Conn.: At no time have there been fungating masses; but there have been lesions as large as English walnuts in these areas, and they have been verrucous. On the anterior part of the left thigh, the skin was perhaps ½ to ¾ inch (1.3 to 2 cm.) thick. The lichenification has appeared since the lesions underwent involution. I am at a loss to explain the condition, because there is no history of the premycotic stage, such as pruritus or other eczematous lesion of long duration. According to the history of this man, he has lost a fortune; he is a highly intelligent man, who now seems nervous, and this condition would fit well in the background for lichen planus, but I cannot make a definite diagnosis.

Granuloma Inguinale. Presented by DR. C. W. O'NEILL, Hartford, Conn.

M. S., a Negress aged 35, in 1930 had a small ulcer on the labia majora. In 1931 two Wassermann reactions of the blood were negative. In 1932 she received radium treatments of the clitoris and perineum. In 1934 she received many injections of bismuth compounds, neoarsphenamine and antimony thioglycollamide. No Donovan bodies were found. In 1935 she had thirty or forty injections of antimony and potassium tartrate, and in 1936 and 1937 she had injections of antimony and potassium tartrate and antimony thioglycollamide and the latter also in 1938. A clitoridectomy was performed in 1938. In 1939 she received seven injections of fuadin (sodium antimony biscatechol sodium disulfonate) intramuscularly and sulfanilamide by mouth for one month. She has received local applications of 2.5 to 25 per cent tannic acid solution, with only subjective relief. Frei tests gave negative reactions.

On the lower half of both labia majora, extending posteriorly on the perineum, in the intergluteal folds, and perianally, there is a red, eroded, granulomatous, edematous, slightly oozing lesion, with a sharply demarcated but thickened border. There is a moderate amount of yellowish white vaginal discharge.

DISCUSSION

DR. C. W. O'NEILL, Hartford, Conn.: The patient has had three varieties of antimony, the salts of which are toxic and may lower the blood pressure and rate of the pulse and result in circulatory collapse. In addition, her veins are disappearing as a result of the numerous injections. One observer has stated that the best way to treat intravenously with antimony salts is to start with the veins in the hand and work up, because of frequent thrombophlebitis. Furthermore, she has been a heavy expense to this community. I think the antimony treatment should be discontinued.

The granuloma has not recurred on the site of an operation for that purpose, and I therefore think a surgical operation could be performed, although the skin is not favorable for it.

Although she wants to do housework, I do not think she should be allowed to do so, as the condition is contagious, unless her employer is told of her condition.

DR. A. LEVENSON, Bridgeport, Conn.: I note that no Donovan bodies have been demonstrated in this particular case in spite of repeated examinations. In view of this fact, I should hesitate to accept the clinical diagnosis of granuloma inguinale. I should also like to know in what percentage of the cases of granuloma inguinale Donovan bodies are not demonstrable.

DR. H. BAILEY, Hartford, Conn.: This patient was presented at the last meeting of the New England Dermatological Society which was held in Hartford, Conn., about four years ago (ARCH. DERMAT. & SYPH. **31**:565 [April] 1935). All the members of that society agreed that the condition was grauloma inguinale. I think Dr. Howard Fox, who also saw the patient, agreed with that diagnosis. As to Donovan bodies, I do not think they are found in 100 per cent of the cases. According to Fox's article (Fox, H.: Granuloma Inguinale: Its Occurrence in the United States, J. A. M. A. **87**: 1785 [Nov. 27] 1926), they are found in about 66 per cent of the cases.

Lichen Planus. Presented by DR. E. M. STANDISH, Hartford, Conn.

M. H., a woman aged 50, consulted me in February 1931 for an eruption of three or four years' duration on the upper and lower eyelids, behind the ears and on the face and chest, which was scaly, erythematous, sharply demarcated and somewhat wrinkled. Although there was no follicular plugging, the appearance suggested lupus erythematosus. A small dose of gold sodium thiosulfate was consequently given. Unknown to me, the patient was taking solution of potassium arsenite, U. S. P. (Fowler's solution) at the same time, and a few days after the injection, dermatitis exfoliativa developed. On May 14, 1931, she was entirely well. She was again seen on Feb. 24, 1939, with scaly slightly raised lesions, with questionable keratotic plugging, on the eyelids and sides of the neck. A diagnosis of seborrheic eczema seemed plausible. Treatment was directed at clearing the scalp. With the local use of mild soothing preparations and roentgen ray treatments, the condition responded but little. It was felt that contacts were possibly playing a part, so all cosmetics, including nail polish, toilet water and soap, were removed; even use of starched clothes was discontinued. In spite of this, other areas appeared that looked more like lichen planus. A bismuth preparation was given weekly by intramuscular injection, without improvement.

The lesions are erythematopapular and sharply demarcated, with exaggerated cutaneous markings. Some seem to contain annular papular lesions which are shiny but not strictly flat. There is also superficial scaling. Lesions on the trunk and other isolated lesions are the same. Areas on the "V" of the chest and sides of the neck present an atrophic-like scaly erythematous eruption of conglomerate lesions, which in some areas show a white, papular, racemose appearance, suggestive of lichen planus sclerosus. There is no true plugging, but much of the area is definitely glistening and shiny.

She has been treated with roentgen rays and local medication, including calamine lotion and cream, boric acid solution, solution of calcium hydroxide U. S. P. and olive oil, and antipruritics, without improvement. She has had calcium gluconate by mouth and intravenously.

DISCUSSION

DR. M. J. STRAUSS, New Haven, Conn.: I saw no histologic evidence of lichen planus. There was no dense infiltration of cells directly below the basal cell layer. I should say that the slide showed simply a chronic inflammatory reaction.

DR. B. APPEL, Boston: Clinically this eruption impressed me as being fairly typical of lichen planus sclerosus et atrophicus. The area is rather extensive and symmetric, the margins are sharply outlined; the skin is smooth, and there are two or three typical flat, pink, shiny papules on the backs of the hands.

DR. J. MULLER, Worchester, Mass.: I cannot agree with the diagnosis of the presenter or of Dr. Appel. I did not see any lesions except one little circle on the wrist which resembled lichen papules. The distribution makes one think of a sensitization dermatitis possibly contact dermatitis or of protein origin.

DR. J. G. DOWNING, Boston: Eight years ago this woman had an attack involving the eyelids, neck, the flexor surfaces of the arms and the legs. The lesions she presents are lichenified. My diagnosis is neurodermatitis.

DR. J. H. SWARTZ, Boston: I agree with Dr. Downing's diagnosis. I think the distribution is typical of neurodermatitis, including the sides of the neck, the flexor surfaces of the elbows and the popliteal spaces. There were a few atrophic scars due to scratching and secondary infection.

DR. E. M. STANDISH, Hartford, Conn.: She had dermatitis in much the same areas that are involved at present. The lesions were fairly small until about six weeks ago. The patient, while on her vacation lost all her money on a train. Within a week the area spread greatly and has remained so to date. Atrophy and the parchment-like appearance have been increasing during the past few weeks. The lesions in the left scapular region have more and more suggested lichen sclerosus et atrophicus. The lesions on the wrists have also changed, particularly the one on the right wrist, which was submitted for microscopic examination. The slide showed definite atrophy.

She has been happy and has a good job, and I know of no reason that might precipitate neurodermatitis. She has been thoroughly contact-tested with everything possible, and everything possibly irritating within her environment has been removed. The only possible finding is the result of the patch test with solution of potassium arsenite U. S. P., which was 2 to 3 plus.

DR. B. APPEL, Boston: I have heard for the second time today the implication of what is commonly referred to as "nervousness" in playing a part in the causation of lichen planus, and I should like to rise in objection to that. It seems to me a bit far fetched to expect such definite somatic pathologic involvement of a structure of the body as a result entirely of mental disturbance. If so-called "nervousness" had anything to do with lichen planus, I should expect a sudden rise in the incidence of lichen planus in all the Czechoslovaks, in all the Polish people, and certainly in the English people right now. I challenge the relation between so-called "nervousness" and lichen planus.

DR. C. W. O'NEILL, Hartford, Conn.: I agree with Dr. Appel. When Dr. Gray was in this country he made the observation that during the World War there was no increase in the incidence of lichen planus in the hospitals at home, in France, in England or at the front among the soldiers. During the last ten years of the depression there should have been some increase in the incidence in this country as well as abroad on the basis of nervous shock.

DR. J. H. SWARTZ, Boston: I am not going to argue about the causation of lichen planus, as I do not know it, but I disagree with Dr. Appel's statement. Dr.

Felix Deutch and others have written a good deal on somatic pathology in relation to psychic disturbance.

Dr. J. Muller, Worcester, Mass.: I will have to disagree with Dr. Appel again. I saw an eruption of lichen planus which developed in a man after he lost his son in an accident. While I have observed a good many cases in which no mental trouble preceded the lichen, I have seen 3 in which the condition followed a substantial monetary loss. So I think there may be something in the relation of nervous trauma to lichen planus.

Dr. E. M. Standish, Hartford, Conn.: I cannot let that statement go unchallenged. One observes numerous cases in which lichen planus fails to improve with different types of therapy. A change of environment and rest gives better results than weeks of therapy. I will not say that change and rest are the principal factor, but I think they form one of the definite factors entering into good results obtained in many cases of lichen planus.

Dr. W. P. Boardman, Boston: I agree with Dr. Standish that we have all observed cases in which the condition followed soon after episodes of mental trauma, and I recall instances in which lichen planus recurred once or twice or even three times after a recurrence of mental disturbance. I do not say that mental disturbance is the cause of lichen planus, but it certainly has some relation.

Dr. V. J. Ryan, Providence, R. I.: I think one ought to bear in mind that the central nervous system and the skin are derived from the same embryonic cells. I agree with Dr. Standish that lichen planus is of nervous origin.

Dr. M. J. Strauss, New Haven, Conn.: Let me point out that the embryonic structure from which the skin and the central nervous system are both derived is the basal cell layer and that the pathologic changes of lichen planus are in the cutis underneath the basal cell layer. My opinion is that nervousness or nervous shock has little to do with the causation of lichen planus. Any one can doubtless recall some personal nervous shock, and until some one presents statistical figures showing that in a goodly number of persons who suffered trauma and shock lichen planus subsequently developed, I am not willing to accept this theory of its causation.

Dr. A. Levenson, Bridgeport, Conn.: I agree with those who contend that a definite neurogenic element precedes and predisposes to the outbreak of lichen planus. This is borne out in many patients whom I have observed. I recall 1 patient in particular, a woman who was active in politics. Every time she came up for election, acute lichen planus developed. She has had five or six recurrent attacks. In most cases, I believe, further neurogenic complications may arise as a result of the intense itching and loss of sleep.

Dr. Loretta J. Cummins, Boston: I have a patient, a woman, who since 1920 has had several relapses or recurrences of lichen planus. She has a responsible executive position in a large company, and when she gets tired and run down or conditions are strenuous typical lichen planus develops.

Lupus Erythematosus Disseminatus Subacutus. Presented by Dr. E. M. Standish, Hartford, Conn.

C. J., a woman aged 32, was in good health previous to her present illness. Late in February 1939 she underwent a severe operation for removal of a wisdom tooth. Shortly thereafter an eruption with burning and itching appeared on her face, scalp and neck. She was treated at first with a strong ointment for impetigo. She then received three injections of gold sodium thiosulfate, beginning with 12.5 mg., and later increased to 25 mg. A bismuth compound (bismudol) was given for three doses with little benefit. After administration of sulfanilamide by mouth, 10 grains (0.65 Gm.) three times daily, she was better for one week; then itching and dissemination returned. She was then treated with bismudol intramuscularly and bismuth oxychloride locally, with no change.

She now has a typical butterfly distribution on the face of dusky red hyperkeratotic areas, with typical follicular plugging. There are isolated lesions on the neck, forehead and cheeks, behind the ears and throughout the scalp. The lesions on the chest, arms and body are undergoing involution.

DISCUSSION

DR. M. J. STRAUSS, New Haven, Conn.: I think the condition in this case is discoid lupus erythematosus rather than lupus erythematosus disseminatus. The latter is a generalized disease with cutaneous manifestations, and I am afraid that frequently the two get mixed up in discussions. I agree that the condition is lupus erythematosus, and it may have been generalized at one time, but it is not lupus erythematosus disseminatus.

DR. E. C. WEISE, Bridgeport, Conn.: I agree with Dr. Strauss.

DR. J. H. SWARTZ, Boston: I do not agree with Dr. Strauss regarding this case. I agree with him that there are cases of discoid lupus erythematosus with extensive involvement that do not fall into the group of the acute disseminated type. I believe this condition is a beginning lupus erythematosus disseminatus superimposed on a previous chronic discoid type rather than generalized chronic lupus erythematosus. The patient has pains in the joints and vascular changes that go with the acute type.

DR. E. M. STANDISH, Hartford, Conn.: This woman was sick enough to make me think she was going to have a fatal attack of acute disseminated lupus erythematosus. She failed to improve with any form of therapy and was growing worse rapidly. Finally another probably infected tooth was removed. Her temperature rose a degree or a degree and a half, and there were lesions practically all over the body. She didn't show any improvement until about two or three weeks after that. To my surprise when she came in today she looked almost well as compared with her appearance in August. She formerly had arthritic symptoms in some of the joints in her fingers, but that is gone. I think the condition is a subacute type of disseminated lupus erythematosus which has perhaps been transformed into a discoid type.

Sarcoid of Boeck. Presented by DR. F. E. KUNKEL (by invitation), Hartford, Conn.

In 1937 Mrs. T. B. aged 51 noticed the gradual onset of lichenoid lesions about the right knee, accompanied with moderately severe itching. In December 1938, after an infection of the upper part of the respiratory tract, small brownish papules slowly developed on the flexor surface of the left wrist, forehead, left ear, upper part of the back, left thigh and leg. These lesions slowly increased in size, a few coalescing to form small plaques. Examination in March 1939 revealed livid, brownish, smooth, sharply defined, semifirm, papular and nodular lesions, which were moderately elevated, varying in diameter from about 2 mm. to 1 cm. and distributed about the aforementioned regions. Occasional small plaques were present on the forehead, left ear, back and left thigh. Smooth, brownish, lichenoid lesions were distributed about the right knee. From March 23 to June 15 weekly intramuscular injections of bismuth subsalicylate were given. On June 22 10 mg. of gold sodium thiosulfate was given intravenously, and 25 mg. was administered intravenously on June 29. During the month of May, the patient received five general ultraviolet irradiations. During March a specimen was removed from the back for microscopic examination, which showed well circumscribed masses of epithelioid cells, a few giant cells and lymphocytes and moderate fibrous tissue hyperplasia. Roentgenograms of the lungs showed septum-like infiltrations radiating from the hilus. There was generalized adenopathy. The spleen and liver were not palpable.

Grouped, macular, brownish lesions are distributed about the extensor surface of the left leg and the flexor surface of the left wrist. Brownish macular lesions

are also present about the right knee and the extensor surface of the right leg. An old bilateral static dermatitis, more pronounced on the right leg, is present. There is also a healed varicose ulcer on the right ankle. Sclerodermatous changes are present in the lower part of both legs, more evident on the right. A keloidal scar is present on the back, where the biopsy specimen was removed.

Intradermal tests with tuberculin gave negative results.

DISCUSSION

DR. B. APPEL, Boston: If this patient has sarcoid it probably is not present on the legs but may be in the small pink soft nodular lesions on the back, one of which was excised for the microscopic examination. In my opinion, the lesions on the legs, particularly the one on the right which has a sclerodermatous appearance, and the pigmented lesions are probably related to venous stasis from the varicose veins, for which she had a previous operation.

DR. F. E. KÙNKEL (by invitation), Hartford, Conn.: The lesions on the leg were papular and nodular at the time I first saw the patient in the Hartford Hospital in March of this year. Of interest in this case is the complete disappearance of all lesions about the back, face, left ear and left knee. I think there is another thing that should be mentioned: In taking specimens for microscopic examination, one should always remember the tendency to develop keloidal changes in a certain number of these cases.

Leukoderma. Presented by DR. C. W. O'NEILL, Hartford, Conn.

For six years Miss O. S., aged 27, has had lesions on her arms, abdomen and legs, which started as small white spots on her legs. Then new lesions developed on her arms and abdomen. They apparently disappear in the winter and reappear on exposure to sunlight in spring and summer.

Scattered over the legs and arms are many variously sized lesions that are depigmented. On the anterior surface of both legs there are treated lesions showing the deposit of pigment. All lesions are macular.

She has been treated with oil of bergamot and ultraviolet irradiation, with no pigmentation; with 10 per cent ammoniated mercury ointment locally and ultraviolet irradiation with pigmentation and gold sodium thiosulfate intravenously and ultraviolet irradiation with pigmentation.

DISCUSSION

DR. B. APPEL, Boston: I am skeptical in this particular case about the relation of the restoration of pigment to the therapy. As a matter of fact, more new areas of vitiligo have developed on the forearms since she has had treatment. It is easy for one to fall into the error of reasoning "post hoc propter hoc." Because in some lesions more pigment develops after the use of a certain form of therapy does not necessarily prove that it did so because of the use of that therapy. I should like to go on record as saying that if pigment did develop in those areas it was propably in spite of and not because of the treatment used.

DR. A. W. CHEEVER, Boston: I am going to raise the question, is the condition all vitiligo? The lesions on the arms certainly are without any question; I can't feel any difference in the texture of the skin. But on the legs it seems to me that there is a decided difference in texture, that the skin is greatly thickened, firm and hard and bound down to the subcutaneous tissue and that there is a sharp line demarcating that thickened area about an inch (2.5 cm.) above the knee, above which the skin feels soft and normal. I would hesitatingly make a diagnosis of two conditions, both of them of pigmentary changes, in the same patient at the same time. I am wondering if the condition is vitiligo in the upper part and scleroderma with depigmentation on the lower part, especially as one of the lesions on the ankle has a violaceous tint, suggesting morphea.

DR. C. W. O'NEILL, Hartford, Conn.: I was hoping that some one would bring out a differentiation between leukoderma and vitiligo. The latest dictum that I have on the difference between leukoderma and vitiligo is that the diseases are not related. Microscopic examination of tissue from the depigmented area in which the diagnosis of true vitiligo has been made, i. e., in which there is hyperpigmentation at the edges of the lesions, will show no oxidase in the tissue by the dopa reaction. If the gray fur of a rabbit is cut off it will be found that the skin is white, and histologic examination shows that no pigment is present. However, if that depigmented skin is subjected to ultraviolet radiation, there will now be pigmentation and the presence of oxidase will be shown by the dopa reaction. Usually when the pigmentation comes into the lesions it is around the hair follicles. In view of the fact that there has been a return of pigment to these lesions, I favor the diagnosis of leukoderma.

I did not hear any discussion about ammoniated mercury. I thought some one would suggest that since there was an ointment base for the mercury the ultraviolet radiation would not affect the lesions because of lack of penetration. The patient did not have any increase in pigment while using oil of bergamot. I should like to have some one discuss further the differentiation between leukoderma and vitiligo. I hope my discussion will cause some dissension. This patient has shown no lesions of leukoderma acquisitum centrifugum, has not had any previous lesions of the skin, inflammatory or otherwise, and has had no hyperpigmentation at the periphery of the lesions.

DR. A. LEVENSON, Bridgeport, Conn.: I am not quite in accord with Dr. O'Neill's differentiation between leukoderma and vitiligo. I have always been under the impression that clinically they cannot be differentiated. In both one sees a loss of pigment with hyperpigmentation at the border of the lesion. The histologic changes show the same picture, and, in addition, the dopa reaction for melanin is negative. The difference between vitiligo and leukoderma is that vitiligo is a primary disease in which the lesions appear spontaneously, with no antecedent history of trauma or an acute or chronic inflammatory cutaneous process. Leukoderma, on the other hand, is always preceded by some history of trauma or exposure to the sun and often follows such chronic cutaneous diseases as psoriasis, exfoliative dermatitis or syphilis. I believe that this is the accepted differentiation between them.

DR. E. C. WEISE, Bridgeport, Conn.: I should like to add a few remarks to Dr. Levenson's statement in that as far as I know it is the consensus that vitiligo is a primary condition and comes without any previous inflammatory change. On the other hand, leukoderma develops after some previous inflammatory change in the skin, for example, at the site of former papules of a secondary syphilid.

DR. J. H. SWARTZ, Boston: Dr. Hollander is here, and I suggest that he be given the privilege to discuss this case.

DR. A. HOLLANDER (by invitation), Springfield, Mass.: I agree with Dr. Swartz, because in my work I found that vitiligo appears only in those cases in which previous lesions are not found. The term leukoderma should be reserved for conditions in which there have been previous lesions, for instance, of syphilis. The term leukoderma aquisitum describes the condition that is a special differentiation between vitiligo and leukoderma. I suggest that the condition in this case is vitiligo.

I never saw any result from mercury, because I use mercury for bleaching the pigment, but I think there is a general opinion that when one says "vitiligo" nothing is known about the causation.

DR. J. H. BLAISDELL, Boston: For my own information I should like to ask Dr. Hollander this question: After an inflammatory change in the skin with the appearance of a white spot, which is called leukoderma, does the term leukoderma imply increased pigmentation surrounding the depigmented area or only a whiteness or blanching of the skin?

DR. A. HOLLANDER (by invitation), Springfield, Mass.: In cases of leukoderma, I understand, there is a white spot in the center of the lesion; and in some cases,

not in all, there is an increase in the pigment surrounding, but in vitiligo such a picture is never seen. One sees only the bleaching in the center of the lesion.

Congenital Fragilitas Crinium. Presented by Dr. E. M. Standish, Hartford, Conn.

The hair of C. G., aged 8 years, has been crinkled since his birth. It straightens out somewhat in the summertime but never grows without breaking off. Most of the scalp is covered with crinkly, wiry hair of varying lengths, approaching normal near the margin of the scalp. There is also considerable alopecia diffusely distributed over the entire scalp.

DISCUSSION

Dr. E. L. Oliver, Boston: The mother told me that the child did not sweat at all, and I wondered what connection there might be between the dry, fragile hair and the absence of sweating. It occurred to me that, although the child does not seem to need thyroid in the ordinary sense, small doses of thyroid might possibly help this condition.

Dr. W. G. Watt, Springfield, Mass.: I should like to ask if in cases of this type any of the members have administered whole pituitary gland orally and, if so, what results were obtained. I have used it in a few cases similar to this and have been pleased with the results.

Juvenile Disseminated Xanthomatosis. Presented by Dr. C. W. O'Neill, Hartford, Conn.

P. G., a child aged 18 months, is presented. At the age of 8 months, when she was teething, lesions developed on her face and forehead. Local application of an ointment made the lesions erythematous. The lesions have been itchy.

Scattered over her face, forehead, and the anterior and posterior parts of her neck, shoulders and arms are discrete pinhead-sized to pea-sized elevated yellowish and yellowish brown, more or less rounded, flat-topped papules. A few similar lesions are on the eyelids. There has not been any familial history of diabetes, xanthelasma or xanthoma tuberosum.

She has been treated with trichloroacetic acid locally, with moderate involution of the lesions.

DISCUSSION

Dr. J. G. Downing, Boston: I recently had a case of generalized xanthomatosis in a baby who showed lesions in the mouth and on the tongue, face, both ears and buttock. The lesions on the buttocks resembled those of nodular syphilis. Many of them, however, were depressed in the center. This child had a pronounced lipemia and has responded well to a fat-free diet. After reviewing an article by Urbach (*Klin. Wchnschr.* **13**:577, 1934), in which there is a classification of the lipoidoses, I believe that the condition of this child fits into the group described by Burger and Grutz.

Dr. C. W. O'Neill, Hartford, Conn.: Apropos of Dr. Downing's remarks, I have not instituted a fat-free diet for this child for the simple reason that this is a growing child, and if a low fat diet is used she will become emaciated. One does not always get an increase in the lipid content of the blood. If the fat content in the food is decreased, the child will draw on the fat content of its body, thereby defeating the purpose of a low fat diet. These patients with xanthomatous cutaneous lesions may have the vital organs also infiltrated with similar lesions. Wile had a patient who showed at autopsy xanthomatous endocarditis. One new approach in therapy has been the use of lipocaic, which is an alcoholic defatted extract of the pancreas. This preparation is also being tried in cases of psoriasis. Disseminate xanthomatosis of childhood persists into adult life. Nevoxanthoendothelioma usually disappears, and there are six lesions or fewer. Sometimes these children also have diabetes insipidus. The concentration of the urine of this child was 1.012, ruling out diabetes insipidus.

Acne Varioliformis. Presented by Dr. M. J. Morrissey and Dr. H. S. Reynolds, Hartford, Conn.

Miss D. P., aged 20, has discrete, papular, acneform lesions involving her forehead and extending into the hair margin, which developed in the summer of 1935. After treatment some of the lesions became confluent. She was first seen on March 15, 1938, at which time she presented the present lesion on the forehead at the hair margin which is 1¾ inches long and 1 inch wide (4.5 by 2.5 cm.), with thickened scar formation and comedos. Discrete papular acneform lesions extended within the hair margin. All the lesions were limited to the forehead, with a few lesions involving the scalp near the hair margin. Previous treatment had consisted of electrodesiccation, one hour of radium (10 mg. plaque), fulguration and three roentgen ray treatments (75 r each). Recent treatment has been with the ultraviolet irradiation with the air-cooled quartz mercury vapor arc lamp, with only slight improvement.

DISCUSSION

Dr. H. S. Reynolds, Hartford, Conn.: I saw this patient about a year ago. She had been under treatment for about three years before that. Today she does not present the same picture that she did a year ago, because in addition to the keloid, she had discrete necrotic lesions twice the size of the head of a pin. These have healed, with slight scarring, but in the larger lesions the keloids still remain, and I should like to know how to treat them.

Dr. B. Appel, Boston: I should like to suggest that in using roentgen ray therapy for keloids one must not be too timid, because if one uses relatively small doses the results will not be satisfactory. Roentgen rays, filtered either with aluminum or with thin copper to the point of five sixths of an erythema dose, the dose in roentgen units depending on the wavelength used, repeated several times, at intervals of from six to ten weeks, are likely to produce a better cosmetic result. If one is going to give a suberythema dose timidly, only once or twice, one must not expect good results.

Dr. E. M. Standish, Hartford, Conn.: I think the treatment was perhaps conservative, because there is no good history of the first treatment given, that of the radium plaque. No one knows, because of the death of the man who gave the treatment, whether or not the radiation was filtered or unfiltered. I believe every one has hesitated on the degree of therapy and has been more conservative. That is the reason why the condition was not treated with a larger dose of roentgen rays.

Dr. B. Appel, Boston: I should like to add that in using roentgen ray therapy as an adjuvant to surgery one should not wait too long, because keloids develop owing to the laying down of fibroblastic cells, which occurs early. One should map out the roentgen ray therapy according to such a plan that the first dose should be given not later than six or seven days after the operation. If one waits until clinical healing has taken place, by that time a considerable amount of fibroblastic tissue has already been laid down in the operative wound, and one is less likely to get a good result.

Dr. F. E. Kunkel (by invitation), Hartford, Conn.: MacKee suggested for the treatment of thick keloids semi-intensive (225 r) treatments given at monthly intervals with 3 mm. of aluminum filtration (MacKee, George M.: X-Rays and Radium in the Treatment of Diseases of the Skin, Philadelphia, Lea & Febiger, 1938, p. 668).

Dr. A. Levenson, Bridgeport, Conn.: I feel that roentgen ray irradiation is contraindicated in this case. The lesion is definitely keloidal and of long standing. The fibrosis is well organized. Such a lesion in my opinion is radioresistant, and further irradiation may result in roentgen ray sequelae. The best method of therapy would be excision of the lesion followed by plastic repair, as Dr. Appel suggested.

Dr. J. Goodman, Boston: It seems to me there is still activity in this lesion, and the question arises as to whether one is going to accomplish anything in treating a keloid when infection is still present. I suggest that this patient be given a

course of injections of staphylococcus toxoid. Activity might be stopped with this treatment. Then roentgen ray irradiation or plastic surgery might be in order.

DR. J. G. DOWNING, Boston: I believe that the lesion this girl originally had was a naevus sebaceus and that what she presents now is the result of treatment, namely, a keloidal scar. The best treatment would be excision and then irradiation or else leaving it alone.

A Case for Diagnosis (Pemphigus Vulgaris, Moniliasis?). Presented by DR. E. M. STANDISH, Hartford, Conn.

Mrs. B. T., aged 48, had a dermatosis which began one year ago with soreness and tenderness of the mouth, followed shortly by dysphagia and bleeding. She showed no systemic reaction at first and was treated for probable thrush. In January 1939 she was seen by a physician in New York and was observed at the Columbia Medical Center. She was admitted to the hospital in February, with a diagnosis of ulcerated stomatitis, for which no cause was discovered. Several Wassermann reactions of the blood, at various times, have been positive and negative. She improved slightly with the elimination of drugs and administration of liver by mouth and intramuscularly. She was discharged to her home but found a varied diet difficult. She lost 20 pounds (9 kg.) during the interval. Six weeks before admission to the Hartford Hospital vesicles and bullae appeared on her legs, for which she received treatment with gentian violet, without benefit. The lesions gradually spread up the legs to the trunk. At this time discharge about several finger nails occurred.

Her lower lip is now eroded and somewhat crusted. The buccal mucosa is also eroded, with irregular superficial ulceration in areas. Bullae are present at intervals on the hard palate. Small bullae without erythematous bases appear in various places on the arms and legs. On the legs, back and arms the lesions have coalesced to form a vesicular, and in some areas serpiginous, border. The centers of several of the lesions are exuding serosanguinous fluid; others are crusted, and some are tanned from treatment. She has paronychia of two finger nails.

The patient received 300,000 U. S. P. units of vitamin D in daily doses, liver and iron by mouth and intramuscularly, a high vitamin diet, tannic acid baths, tanning with temporary relief, potassium permanganate baths, paste of zinc oxide with salicylic acid (modified Lassar's paste), zinc oxide in olive oil, and boric acid solution wet dressings.

DISCUSSION

DR. F. E. KUNKEL (by invitation), Hartford, Conn.: I should like to get the opinion of the society as regards the treatment of pemphigus with germanin.

DR. W. G. WATT, Springfield, Mass.: I had the opportunity of taking part in the treatment of a patient with pemphigus with germanin. The patient died later, and the germanin had accomplished nothing.

DR. J. MULLER, Worcester, Mass.: I used germanin in 3 cases, without benefit. Among the few patients that I have treated, excellent results have been obtained with vitamin D in 1 patient with pemphigus vulgaris. The patient has been taking 20,000 U. S. P. units a day for several years, which seems to be her maintenance dose. It costs her a great deal of money for the medicine; on several occasions she tried to do without it, and always her condition became worse. I have not seen her now for nearly two years.

Dermatitis Herpetiformis. Presented by DR. M. J. MORRISSEY and DR. H. S. REYNOLDS, Hartford, Conn.

P. S., a Polish man aged 50, has had a dermatosis for one year, which began with an intense itching and with small discrete erythematous and vesicular lesions on the extremities and torso, which later became more widespread with more intense itching. He was first seen on July 31, 1939, at which time he presented widespread discrete macular and vesicular lesions, with marks and bleeding lesions from scratching. Later there was extension to the neck and scalp.

He has had two roentgen ray treatments (¼ skin unit, 75 r) to date, with topical applications, and has received solution of potassium arsenite U. S. P. three times a day by mouth, with some improvement.

DISCUSSION

DR. J. H. SWARTZ, Boston: I should like to suggest a diagnosis of lichen planus in this case. Examination showed definite flat, angular violaceous papules on the flexor aspects of the forearms and some definite annular lesions. I believe that a biopsy would be advisable. There was pigmentation but no grouping and no herpetiform eruption.

DR. M. J. MORRISSEY, Hartford, Conn.: Dr. Reynolds and I saw the patient about two months ago for the first time, and at that time his scapulas, sacrum, elbows and knees were covered with discrete vesicles, which resembled dermatitis venenata. He had many remissions and exacerbations during that time. In the last two weeks nearly all the vesicles have disappeared excepting one or two on the shoulders, and the picture is absolutely changed. When we saw him first there was a definite grouping of the lesions, particularly around the scapulas and the sacrum, but now nearly the whole body has become so involved that one cannot point to any particular grouping of the lesions. We were rather inclined this morning to change the diagnosis when we saw the condition had changed from the grouped lesions with intense itching and burning to lichen-planus–like residual lesions wherever there was a lesion before, with even the violaceous sheen to it. However, considering the history we still believe that the condition is dermatitis herpetiformis and that the treatment given him has changed the picture from what it was a week or so ago. I do not believe lichen planus vesiculosus would show so many lesions.

Eczema Vaccinatum. Presented by DR. H. BAILEY, Hartford, Conn.

C. Y., aged 24, has had eczema (atopic dermatitis) of the face, neck, chest and extremities for the past six years. A dermatosis developed five days previous to entrance in the hospital on Dec. 23, 1938. He stated that he had come in contact with his child, who had been vaccinated two and a half weeks previously.

The scalp showed many pustular and crusted lesions in the occipital region. There were many discrete and confluent umbilicated pustular lesions on the face, more pronounced on the forehead and upper parts of the cheeks. There was edema of the left upper eyelid, with a few scattered umbilicated pustules. There were a few scattered similar lesions on the neck and chest. The upper extremities, especially the flexor surfaces, showed many umbilicated pustules and crusts. The right hand and elbow were swollen, reddened and covered with many umbilicated pustules. The palms were free of lesions. The inguinal and axillary lymph nodes were enlarged. The lower extremities, predominantly in the popliteal areas, were covered with similar lesions. At present he shows a few pitted scars on the left side of the forehead and in front of the left ear. The eczema (atopic dermatitis) of the face, neck, chest and extremities is still present.

He has had local applications, ammoniated mercury 3½ per cent and ultraviolet irradiation.

DISCUSSION

DR. H. BAILEY, Hartford, Conn.: This case was presented not solely because of the atopic dermatitis but because of the eczema vaccinatum, which was proved. Various diagnoses were made, ranging from smallpox to chickenpox. The eruption was of a pustular, umbilicated type and present over the areas of eczema.

DR. E. M. STANDISH, Hartford, Conn.: I should like to corroborate Dr. Bailey's diagnosis of vaccinia.

DR. W. T. GARFIELD, Boston: I had a case not long ago at the Boston City Hospital in which in a child with eczema, vaccinia lesions developed on the arms principally. Other children in the family were vaccinated about seventeen days

previous to the beginning of the eruption. This child was sent to the South Department for Infectious Diseases of the Boston City Hospital, and under treatment there it got well. The lesions were examined for smallpox virus, which was definitely found in this case.

DR. E. L. OLIVER, Boston: I should like to add that during a smallpox epidemic I cannot see how these cases can be differentiated from true smallpox. The child I saw recently certainly had an eruption which looked like smallpox. Vaccinia of this type, I understand, has a mortality of over 30 per cent.

DR. J. H. SWARTZ, Boston: I do not remember who brought out the fact that in treating eczema in children with crude coal tar the possibility of eczema vaccinatum is more likely.

DR. H. BAILEY, Hartford, Conn.: There was no coal tar used at all. In fact, the patient had not had treatment for several months previous to that time. A question of nomenclature arises in this case, and this is brought out very nicely in an article by Tedder (Tedder, J. W.: Eczema Vaccinatum, ARCH. DERMAT. & SYPH. **34:** 1008 [Dec.] 1936). He differentiates eczema vaccinatum from general vaccinia. I think if it is a question of nomenclature the condition in which there is eczema should be called eczema vaccinatum, and those in which there are no other lesions of the skin should be called general vaccinia.

Lichen Planus Bullosus. Presented by DR. C. W. O'NEILL, Hartford, Conn.

O. M., a man aged 40, has had a duodenal ulcer for four years and is being treated at the Veterans' Administration Facility at Newington, Conn., with a Sippy diet. His present eruption started six months ago as "water blisters" on his legs and has been itchy. There has been a gradual spread of lesions to the arms, wrists, abdomen, penis, legs and thighs.

There are many pinhead-sized, flat-topped, angulated, erythematous to violaceous, shiny, discrete, linear papules, symmetrically distributed on the flexor surfaces of the wrists and forearms. On the abdomen and thighs there are similar pea-sized lesions tending to coalesce. There are a few scattered lesions on the chest. Similar pinhead-sized lesions are on the penis. On the legs there are many similar coalesced pea-sized excoriated lesions, with some bullae and crusting present. The buccal mucosa shows ill defined whitish patches. There is moderate scaling between the toes.

Treatment has consisted of application of lotions and intramuscular injections of a bismuth compound.

Lupus Vulgaris; Roentgen Ray Dermatitis; Tubular Repair. Presented by DR. C. W. O'NEILL, Hartford, Conn.

Since 1917, when he was in the army, J. S., aged 47, has had dermatosis involving the anterior surface of his neck, the sides of the neck, extending retroauricularly, the right ear, the right buttock and the right thigh posteriorly and laterally. At the onset the lesions involved the right side of the neck and the buttocks. He was hospitalized in France, for eight months in Scotland, at the Veterans' Administration Facility, Newington, Conn., in the Veterans' Administration Facility in New York, and at the Hartford Municipal Hospital in 1935 for removal of epithelioma of the right side of the neck and a pinch graft and on Aug. 24, 1939, for a tube graft in the left side of his neck and roentgen ray dermatitis. He has had much roentgen ray irradiation, with secondary sequelae.

The right ear is thinned, scaly and erythematous, and piled-up serum debris is present on the tragus. Retroauricularly on the right side and extending down anteriorly across the neck to the left ear there is a large continuous plaque showing scarring, atrophy, telangiectasis, dryness and thinned-out and pigmented skin. In the middle portion of the neck there is an ulcer with accumulated serum debris. The left side of the neck is bandaged. There is a large plaque involving all of the right buttock and extending down the thigh posteriorly and laterally. On the buttock

there is a 3 inch (7.5 cm.) area showing depigmentation, pigmentation, thinning, scarring, moderate scaling and atrophy. The remainder of the buttock and thigh shows redness, scaling and thickening.

On Jan. 14, 1935, the report of the excised epithelioma was epidermoid carcinoma grade 2.

On Sept. 26, 1939, microscopic examination of the buttocks showed inflammatory tissue, probably tuberculous.

Bullous Lichen Planus. Presented by DR. R. A. MCDONNELL, New Haven, Conn.

A. H. B., a stationary engineer, was first seen on Aug. 30, 1907. He had had for two months an extensive eruption of lichen planus. By November 30 his condition had cleared. He was next seen on Sept. 15, 1910, for typical lichen planus confined to the legs, while the previous attack had been chiefly of the arms. On Jan. 18, 1911, the stains were fading, and there was no activity. On March 18, 1913, he showed a fresh papular attack, mostly on the legs, and in places the condition appeared psoriasiform. Within two months the eruption cleared. In March 1917, he had a vesiculopapular eruption of the legs and forearms, which at that time was distinctly eczematous in appearance. He was clear on July 7. He was next seen on Jan. 1, 1930, when he stated that he had had a light attack three years before which spontaneously cleared. The eruption at that time was of one week's duration and was again psoriasiform in appearance and chiefly confined to his legs. It cleared in two months.

The present eruption began about Aug. 1, 1939, as typical lichen planus, chiefly on the legs, with some on his forearms. On October 12 he presented this unusual phase of numerous superficial bullae and vesicles, extending from the hips to the ankles, and for this reason he is being presented today.

Creeping Eruption Due to Ancylostomiasis. Presented by DR. R. A. MCDONNELL, New Haven, Conn.

A. P., aged 82, of Indian and American stock, entered the dermatologic clinic of the New Haven Hospital on Oct. 9, 1939. He complained of an eruption appearing during his visit to Florida this past summer. It is confined to the legs and is steadily becoming worse. His chief complaint has been intermittent itching. On examination he showed numerous gyrate burrows within the skin, in places reaching a total length of 10 cm. There were numerous excoriations. The diagnosis was creeping eruption. It was later confirmed by the finding of the larva of a nematode. He was given a placebo and referred to the medical clinic for a complete physical examination.

There was a history of syphilis. Symptoms of prostatic enlargement were found. The heart was enlarged and had an aortic diastolic murmur compatible with some aortic insufficiency. Arteriosclerosis with hypertension was present.

On the patient's return to the dermatologic clinic the two areas were treated with ethyl chloride spray, with the result that there was an immediate quieting of the pruritus and within forty-eight hours a decided decrease in the activity of the lesions treated.

DISCUSSION

DR. E. L. OLIVER, Boston: I observed an interesting case two years ago. A young man went to Venezuela on his honeymoon and sat on the sand at the beach; a few weeks later he was covered with linear lesions, especially on his buttocks and also on his legs and ankles. He must have had at least a hundred lesions. I saw him only twice. I froze all the lesions with ethyl chloride, many of them at both ends because I could not tell which end contained the parasite. He came back in about ten days, and there were only eight lesions left; about ninety-two had completely disappeared. I treated those eight lesions, and I never saw him again, but I understand from his friends that he completely recovered after those two treatments.

DR. F. E. KUNKEL (by invitation), Hartford, Conn.: I reported an extensive case of creeping eruption in 1938 (*Delaware State M. J.* **10:** 123 [June] 1938). It was the first case reported in which the condition originated in the state of Delaware. The patient had numerous lesions distributed over the left thigh, left hip, lower left quadrant of the abdomen and left arm. In the treatment, it is important to first treat secondary pyogenic infections if present. I did not refrigerate the lesions for as long a period as Kirby-Smith and Shelmire advise (Kirby-Smith, J. L.: The Treatment of Creeping Eruption, *South. M. J.* **28:** 999, 1935). The lesions were refrigerated for not more than thirty seconds with an ethyl chloride spray, and I was able to cure practically all of them. A few that I did not cure I refrigerated with solid carbon dioxide for five seconds.

DR. R. A. MCDONNELL, New Haven, Conn.: This particular condition was contracted in Miami, and the nematode larva was responsible. One of the seniors in the medical school is studying the case. He is attempting to pass the larva into a cat and may get some good specimens. Of course, the parasite is one of the three different types which cause this eruption; the Florida type is generally the Ancylostoma braziliense. Incidentally, this man has only had a simple cream of water and starch, and the lesions he scratched have cleared spontaneously with that cream. He has not a fourth of the lesions now that he did have.

Cutaneous and Subcutaneous Calcinosis. Scleroderma (Right Cheek)? Presented by DR. H. BAILEY, Hartford, Conn.

J. S., a boy aged 12 years, fell from a veranda and injured his leg when 2 years of age. After this injury he suffered from frequent nosebleeds for one year. He first entered the hospital in August 1931, at the age of 4, with swelling and pain of both legs and knees of two years' duration. He entered the hospital again the following November with a tender swelling of the left leg below the knee. This was incised and drained. At that time he also showed a reddish area on the right cheek, about 2 inches (5 cm.) in diameter. Roentgen ray examination showed calcium deposits in the upper and lower extremities. In January 1933 he entered the hospital with palpable masses of the left arm. On December 9 he was admitted to the Hartford Municipal Hospital, Department of Communicable Diseases, with pharyngeal diphtheria. In March 1935 he reentered the hospital with cellulitis of the right leg, and in March 1938, with an infection in the left groin. He was last seen in the outpatient department of the Hartford Municipal Hospital on Oct. 5, 1939.

He now presents small erythematous areas on the lateral surface of the right upper and lower eyelids. On the right cheek in front of the ear are two erythematous scaly, slightly thickened patches measuring 1½ by 2 inches (3.8 by 5 cm.). There is also an erythematous area on the right side of the chin. On the extensor surfaces of both elbows are pinpoint-sized to pea-sized whitish nodules. Subcutaneous nodules can also be felt along the right forearm. The lower extremities present various-sized cutaneous and subcutaneous nodes, most prevalent on the crest of the ilium, right thigh and knee joint and on the anterior surface of the left leg. There is a flexion deformity of the right knee joint, with a hard induration of the hamstring muscles in the popliteal area. The left leg is bigger than the right in the middle portion. Scars of previous lesions are present on both upper and lower extremities.

DISCUSSION

DR. E. L. OLIVER, Boston: I think the lesion above the right knee is much more characteristic of scleroderma than those on the face. I have never seen a localized scleroderma with as much redness as those lesions on the face; and furthermore, there was not much hardness that I could make out; they were barely palpable.

Psoriasis. Bacterid? Dermatophytosis? With Dermatophytide? Presented by DR. C. W. O'NEILL, Hartford, Conn.

Two and a half years ago lesions developed on the hands of E. E., aged 44, and then one month later they spread to the feet. At onset there were a few lesions on

the hands. After the patient had bowled and used towels of the bowling alley, four or five days later, the hands became entirely involved. The lesions started as vesicles. The interdigital spaces also had cellular debris and vesicles. Over the sternum there was a moist, erythematous area 6 inches (15 cm.) long. There was much dry scaling of the scalp. The scalp and the sternal lesions cleared. The hands and feet have had exacerbations and remissions.

Involving each entire palm there is a continuous plaque of sharply demarcated, finely scaly, erythematous tender skin. The lesions extend up the fingers and involve the webs and the anterior surfaces of half the fingers. On the soles there are medially a few scattered pea-sized to dime-sized scaly dry lesions. Extending between all the toes from the anterior surface to the plantar surfaces there is piled-up scaly debris, with much desquamation on the plantar surface and with a sharply demarcated border.

Powders, lotions, emulsions, ointments and ultraviolet therapy have been employed. A low fat diet, vitamin therapy and special diet have been used. Microscopic examination showed psoriasis.

DISCUSSION

DR. E. C. WEISE, Bridgeport, Conn.: I suggest the diagnosis of ordinary palmar and plantar psoriasis. There is one typical psoriatic lesion in the left antecubital space.

DR. J. G. DOWNING, Boston: My diagnosis is pustular psoriasis. I think the use of a vaccine made from cultures from the tonsils, plus fairly large doses of arsenic, might be of value in this case. I have tried this therapy in 2 cases, with fairly good results. The last case showed a pneumococcus culture from the tonsil. One does not always get pneumococcus from the throat; in some cases one gets a combination of staphylococcus and streptococcus.

Leukoderma Following Dress Shield Dermatitis. Presented by DR. H. BAILEY, Hartford, Conn.

In October 1938, A. F., a Negress aged 47, while wearing a pair of new dress shields one afternoon experienced a burning and itching sensation in both axillas. That same night she noticed that the skin in the axillary regions was red, irritated and slightly swollen. A month later the eruption subsided, after which dipigmentation occurred.

There are two depigmented areas in the axillas, which extend to the under surfaces of the arms, sides of the chest and back. The depigmented areas are symmetric and follow the outline of the dress shields. For several months thereafter the depigmented areas increased in size. Patch tests were negative to cloth, cloth and rubber and rubber alone. However, when the patient wore the dress shields a positive reaction was obtained.

SAN FRANCISCO DERMATOLOGICAL SOCIETY

MERLIN TREVOR-ROPER MAYNARD, M.D., *President*

H. V. ALLINGTON, M.D., *Secretary*

Nov. 17, 1939

Delayed Positive Reaction to Patch Test with Nupercaine Ointment. Presented by DR. GEORGE V. KULCHAR.

L. L., a white woman aged 64, is presented. Areas of erythema developed after roentgen ray treatment of a basal cell epithelioma in the left infraorbital region on Sept. 28, 1939. Nupercaine ointment was applied. Two days later acute dermatitis

developed at the site of the applications. A patch test was made with nupercaine ointment on the right arm. The reaction was negative at forty-eight hours and remained negative until November 16, forty-five days later, when a strongly positive eczematoid reaction appeared at the site of the patch test and is still present. The dermatitis has disappeared, but a slight roentgen ray reaction still remains.

DISCUSSION

DR. H. J. TEMPLETON, Oakland: Dr. Alderson, did you not present here some time ago a patient in whom a granulomatous process developed after the use of nupercaine ointment?

DR. HARRY E. ALDERSON: Yes; I presented a patient with rhinoscleroma (ARCH. DERMAT. & SYPH. **36**:1018 [Nov.] 1937), who received heavy roentgen ray therapy. The roentgenologist without my knowledge gave her nupercaine ointment, and application of this resulted in violent dermatitis venenata over most of both cheeks. After that, the rhinoscleroma spread to that area. The patient was also presented at a meeting of the American Dermatological Association (ARCH. DERMAT. & SYPH. **39**:1057 [June] 1939).

I cannot account for the greatly delayed reaction at the site of the patch test made over a month ago. The positive reaction, which appeared just a few days ago, is definite. I should like at this time to refer to the occurrence of dermatitis in many cases after the application of nupercaine ointment. Unfortunately, this ointment seems to be used indiscriminately by many physicians, probably because it is so well advertised. It is a favorite with some roentgenologists, who seem to think it will modify an expected reaction after roentgen ray treatment. I have observed several cases of severe spreading dermatitis venenata due to such applications.

DR. ERVIN EPSTEIN, Oakland: I should like to know how Dr. Kulchar can explain the reaction occurring forty-five days after the application of nupercaine. As far as I know, the development of a positive reaction to a patch test is dependent on the interaction of a hapten on an antigen with sensitized cells. Of course, it might require six and a half weeks for the sensitization of a given group of cells to become manifest. However, it would still be necessary for some of the sensitizing agent to be present at the time of the reaction. It seems doubtful if any of the nupercaine could have remained in the patient's clothes or skin for forty-five days.

DR. H. V. ALLINGTON, Oakland: The incubation period in poison oak dermatitis can be at least three weeks. It seems possible to me that even longer latent periods, as in this case, are easily possible.

DR. HIRAM E. MILLER: I should like to discuss this case from two standpoints. First, a negative reaction to a patch test, when the test is made at a distance from the area of dermatitis, does not rule out the tested substance as being the cause of the dermatitis. Secondly, the long-delayed so-called positive reaction to a patch test in this patient may be explained on the basis of a subsequent exposure to the substance used in the test and a reactivation of the site of the previous test which formerly gave a subclinical reaction.

DR. GEORGE V. KULCHAR: A delayed reaction to a patch test is not uncommon. One point which appeared to me to be against this diagnosis was that the reaction would be expected to occur at the site where nupercaine was applied by the radiologist. I applied the patch test in another area.

Lichen Spinulosus (Acquired). Presented by DR. MERLIN TREVOR-ROPER MAYNARD, San Jose.

R. F., a boy aged 5 years and 4 months, was first seen on Nov. 10, 1939. One week earlier the mother noticed a roughening of the boy's skin, particularly at the back of the neck, and a scaling in the scalp. She states that a barber in cutting the boy's hair said that the skin looked dirty.

The patient was seen three years ago for ringworm of the scalp, which cleared in six weeks. He had been well otherwise. He was vaccinated for smallpox and was given an injection of toxoid for diphtheria a week ago. The child's diet has contained sufficient butter and egg yolk for a normal vitamin A supply but an insufficient amount of fresh milk. Fat, with the exception of butter, is somewhat low.

This patient is the second one with this interesting condition that I have presented. The first, a boy aged 9 years, was shown in 1932 (ARCH. DERMAT. & SYPH. **26:** 751 [Oct.] 1932). The condition was of one and one half months' duration and was more severe. Itching, which was present, stopped after three injections of the patient's own blood.

DISCUSSION

DR. GEORGE V. KULCHAR: I should like to suggest a diagnosis of keratosis follicularis contagiosa. This occurs in children.

DR. HIRAM E. MILLER: There was one unusual thing about this case. The keratotic lesions were not grouped. The condition is a diffuse process and is probably a follicular keratotic reaction to some toxin and not the disease entity lichen spinulosus.

DR. H. J. TEMPLETON, Oakland: I think the condition is a toxic reaction of the follicular apparatus resulting from the giving of diphtheria toxoid or from vaccination.

DR. HARRY E. ALDERSON: How about avitaminosis in this case?

DR. MERLIN TREVOR-ROPER MAYNARD, San Jose: The patient is getting vitamin A now.

DR. O. P. DIEDERICH, Fresno: I noticed that the skin was particularly dry. Such a condition is usually associated with dryness of the skin.

A Case for Diagnosis (Necrobiosis Lipoidica Diabeticorum?). Presented by DR. L. R. TAUSSIG.

S. R., a white woman aged 70, was first seen at the University of California Clinic in 1937. At that time a diagnosis of gallstones was made. She was given a fat-free diet and was not seen again until January 1939, when she came to the clinic complaining of painful nodules on the legs, hands, arms and sclera of the left eye. The nodules had persisted for two months. The affected areas showed a patchy induration with purplish center and faintly erythematous border. They were tender to touch, pruritic and burning. A diagnosis of diabetes mellitus and necrobiosis lipoidica diabeticorum was tentatively made. The diabetes was controlled by diet. The tender nodules have continued to appear. They regress in about six weeks.

In 1937 the urine showed no sugar. In February 1939 a dextrose tolerance test showed the following result:

After Administration of Dextrose	Milligrams per Hundred Cubic Centimeters
One-half hour	175
One hour	235
Two hours	238
Three hours	202

In October 1939 the fasting blood sugar was 143 mg. per hundred cubic centimeters; the plasma cholesterol was 187 mg. per hundred cubic centimeters. Microscopic study showed obliterating arteritis and fat necrosis compatible with the diagnosis given.

DISCUSSION

DR. N. N. EPSTEIN: I have seen this patient on several occasions. I believe she has a form of panniculitis rather than necrobiosis lipoidica diabeticorum.

DR. ERVIN EPSTEIN, Oakland: I agree with Dr. N. N. Epstein. It is not usual for the lesions of necrobiosis lipoidica diabeticorum to appear and disappear as this woman says her lesions have done. Also, the lack of yellowish coloration is against that diagnosis.

DR. ARNE E. INGELS: I discussed the sections with Dr. Taussig. They show a lobulated appearance suggestive of sarcoid. There was no tubercle formation or central necrosis. There was a spongy appearance of some of the cells, which were foam cells presumably. Also, the presence of giant cells and the sudan-stained sections, which showed a large amount of fat, seemed to contradict a diagnosis of necrobiosis, in which little fat is found and there is scar formation. It would be unlikely that her mild, controlled diabetes should be the etiologic factor.

DR. L. R. TAUSSIG: This case does not clinically resemble those of necrobiosis lipoidica diabeticorum previously described. The sections show areas of necrobiosis, and in the fat stain there are some inspissated globules of fat in the tissue. I believe that the conditions is related to necrobiosis lipoidica and that it is associated etiologically with the patient's mild diabetes. The nodules first appeared at the time of the beginning of the diabetes. However, further study may make this diagnosis untenable. At the present time the patient is being studied in the diabetic clinic, and perhaps more will be known when the studies are completed. The histologic changes do not correspond with those of sarcoid.

A Case for Diagnosis (Pigmentation of Skin of Forehead). Presented by DR. C. J. LUNSFORD, Oakland.

A. A., a man aged 20, seen at the E. V. Cowell Memorial Hospital at Berkeley, first noticed a discoloration of the skin of his forehead about the middle of August 1939. This has deepened in color and spread since that time. During the last two weeks new areas have developed. There are no subjective symptoms. The patient states that in the mornings when he first arises the color is much faded, as compared with the color seen the rest of the day. There is no history of drug intake. He has not been ill except for an occasional cold for which, several years ago, he sprayed his nose on a few occasions with a preparation the contents of which are unknown. There is no history of injury.

He presents on the right side of the forehead patches of dark brown pigmentation which are more or less in the pattern of vertical streaks about ¼ inch (0.6 cm.) in width. They are well circumscribed, macular, not scaly and without signs of inflammatory reaction. The lesion of deepest color is the oldest and is near the midline. There is a slight fading on pressure, which can probably be accounted for by the expulsion of blood from the normal superficial capillaries. A biopsy specimen was taken on November 10. The microscopic picture is that of a chronic inflammatory reaction. The pigment, stained with prussian blue, is seen to be hemosiderin.

DISCUSSION

DR. EUGEN OSTWALD (by invitation): I have observed a number of cases of hyperpigmentations that developed after use of cheap eau de cologne, bergamot oil or even benzine during exposure to the sun. I am speaking of the so-called berlock dermatitis. The lesions never showed signs of dermatitis but only hyperpigmentation. They developed in the course of a few days to a few months when the cheap perfumes were used while the persons were exposed to strong sunlight. The configuration of the lesions suggests this diagnosis.

DR. MERLIN TREVOR-ROPER MAYNARD, San Jose: Did the lesions occur after a single application of the perfume or after many?

DR. EUGEN OSTWALD (by invitation): Sometimes the lesions occurred after only a single application.

DR. ARNE E. INGELS: I have recently seen 2 patients, 1 with Dr. Alderson, who had lesions on the lip attributed to the smoking of cigarets. The lesions raised a

question of possible melanoma. Both patients were women. One confessed to smoking a cheap Mexican cigaret. Chronic irritation of some kind might well be a factor in this case.

DR. L. R. TAUSSIG: I am certain that this case is not one of berlock dermatitis. The section shows that the pigment is hemosiderin. That, of course, rules out an external factor as the cause of the pigmentation. I had in mind the possibility of Kaposi's sarcoma.

DR. HIRAM E. MILLER: I am inclined to believe that the condition in this case is a trophic disturbance. The patient had a blow over the area, not severe, and he associates the lesion with the blow.

DR. C. J. LUNSFORD, Oakland: I agree with Dr. Taussig that the presence of hemosiderin rules out the other diagnoses suggested.

Recurrent Erysipelas of the Face. Presented by DR. N. N. EPSTEIN.

F. P., an Italian girl aged 18, has had for the past eight years frequently recurring infections in the upper part of the respiratory tract, with chills and fever, swelling of the submaxillary glands and otitis media on the left side. On several occasions incision of the glands of the neck yielded purulent discharge. At the age of 10 the patient had an appendectomy; at the age of 12, a tonsillectomy. About two years ago the left side of the face became swollen, tender and red during an infection of the upper part of the respiratory tract. For the past six months swelling of the face, fever, swollen glands and pains in the arms and legs have occurred about every two or three weeks, lasting from one to ten days.

The patient first entered the University of California Hospital in October 1939, at which time she had an attack similar to the present one. Her temperature was 101.3 F. (38.5 C.). Administration of sulfanilamide caused improvement in about nine days, and the patient was discharged as cured. Physical examination and chemical examination of the blood revealed nothing significant. Culture of the throat showed nonhemolytic Staphylococcus albus and Streptococcus viridans. Culture of material from the left ear showed nonhemolytic Staph. albus.

The patient again entered the hospital on November 16, with a recurrence of the infection. Her temperature was 104 F. (40 C.) at entry. The left side of the face was swollen, hot, tender and slightly erythematous. The skin felt brawny. A definite margin could be palpated in some areas. There were no other cutaneous lesions save two small fibromas, one on the left thigh and one on the right calf.

DISCUSSION

DR. H. J. TEMPLETON, Oakland: This type of case interests me a great deal. I have seen patients who have given the history of having had as many as twenty-five or thirty recurring attacks of erysipelas of varying severity, some with high fever, general malaise and vomiting. In several cases I have had good luck with long-continued desensitization or immunization with Streptococcus erysipelatis vaccine. I think that sulfanilamide would be better, however, for single attacks.

DR. GEORGE V. KULCHAR: I think that probably the use of erysipelas vaccine is nonspecific. Even stock streptococcus and stock staphylococcus vaccines can be used similarly with good results.

DR. HIRAM E. MILLER: I have always had the most success by treating the fissures at the angles of the nostrils, thereby removing the portal of entry for the infection. The same method applies to recurrent erysipelatoid lesions on the legs, as the infection enters through fissures in the toe webs.

DR. N. N. EPSTEIN: This case may not be one of true erysipelas. It is one of a recurrent adenitis with an associated erysipelas-like cellulitis.

I have never seen a patient with recurrent erysipelas permanently cured. I have treated 2 such patients with roentgen rays. The individual attacks have cleared, and the patients have remained well for from months to years, but recurrences eventually have taken place.

A Case for Diagnosis. Presented by Dr. N. N. Epstein.

F. E., a white woman aged 58, first noticed grayish scaly areas on the vermilion border of the lips about eight months ago. There were slight burning and itching about the same time. Whitish plaques then appeared on the anterior surface of the tongue. At present there are also reticular lesions on the buccal surface of each cheek. The results of physical examination are otherwise negative.

The Wassermann and Kahn reactions were negative. Urinalysis gave negative results. A blood count showed slight secondary anemia.

The patient has received seven intramuscular injections of bismuth salicylate in oil, 1 cc. each, with some improvement.

DISCUSSION

Dr. Charles Albert Shumate: I believe a contact dermatitis should be thought of.

Dr. H. V. Allington, Oakland: I think the lesions are those of lichen planus.

(Dr. H. J. Tempeton, Oakland, and Dr. Merlin Trevor-Roper Maynard, San Jose, agreed with the diagnosis of lichen planus.)

Dr. Harry E. Alderson: Have any of these lesions been irritated or ulcerated?

Dr. N. N. Epstein: They have been irritated but not ulcerated.

Dr. Harry E. Alderson: There is no possibility of galvanic action there?

Dr. N. N. Epstein: I do not believe so. The lesions have not changed except to show slight improvement. This seemed to me to make a diagnosis of contact dermatitis unlikely.

Tinea Cruris and Corporis, Probably Due to Trichophyton Purpureum. Presented by Dr. Ervin Epstein, Oakland.

An eruption developed in the groins of M. H., a white man aged 60, two years ago. This rapidly spread to involve the perineum, natal cleft and sacrum. He has been receiving care since January 1938, treatment consisting of the application of various parasiticides. While there has been intermittent benefit, the patient is little improved. He presents a typical dull red circinate eruption of the groins extending into the natal cleft. Just superior to this cleft the eruption fans out into a semicircle, which is larger than a palm. Most of the activity is in the periphery of this lesion and consists of an unbroken margin of erythema and scaling. The lesions within its borders are red, with some tendency toward a follicular grouping, although this is irregular. Scrapings from the border still show long, nonbranching myceliums. Cultures have been made and growth started, but as yet this is not far enough advanced to allow for definite identification of the pathogenic fungus involved.

DISCUSSION

Dr. Harry E. Alderson: I do not think there is any question about the clinical diagnosis. As to the therapy, I suggest rubbing lemon juice into the eruption three times a day and following that with a powder containing salicylic acid and boric acid.

Dr. C. J. Lunsford, Oakland: There is an induration about the periphery of those lesions. I remember 1 case of proved fungous infection of the feet in which I finally made a Wassermann test and found a positive reaction. After antisyphilitic treatment for two or three weeks, the tinea was cured without difficulty. I was wondering whether there might be any underlying disease that has made the condition resistant to treatment in this case.

Dr. Esteban Reyes (by invitation), San Salvador: I have observed some cases of this type in which the condition was resistant to treatment. I use a 20 or 30 per cent solution of iodine three times a day and continue this treatment for weeks.

DR. ARNE E. INGELS: I think it would be well to learn whether Trichophyton purpureum is the causative factor in this case. In some cases the condition has responded to the use of trichophytin.

DR. HIRAM E. MILLER: The treatment of ringworm infections with trichophytin was discussed at the Philadelphia meeting of the American Academy of Dermatology (not published), and it was the general consensus that trichophytin was not of particular value in treatment. The ringworm infection may be aggravated by the vaccine and subsequently cured by soothing remedies.

DR. HARRY E. ALDERSON: I was wondering whether the high percentage of iodine used by Dr. Reyes was possible because patients perspire freely in his country. In the Orient a 25 per cent alcholic solution of salicylic acid can be used without producing the irritation one would get in this country.

DR. ERVIN EPSTEIN, Oakland: In the case of this patient the Wassermann reaction was negative, although there might be some other underlying condition. At the last meeting the point was brought up that these conditions are resistant to treatment. I think it is interesting that a probable clinical diagnosis can be made of the causative organism by the clinical appearance and by the resistance of the lesion to treatment. This organism is certainly not an uncommon pathogen in this part of the country.

Epidermolysis Bullosa (Acquired). Presented by DR. ERVIN EPSTEIN, Oakland.

D. K., a white man aged 50, was first observed on Oct. 29, 1939, because of an eruption involving his hands. The lesions had first appeared four or five weeks previously while he was gardening. He stated that he had not suffered previously from a similar condition. He stated that there was no consanguinity of his parents.

The patient presented a number of bullous, crusted and superficially ulcerated lesions involving both hands. The sides and dorsa were more extensively affected, although the palms were also involved. When questioned specifically, the patient admitted that perhaps trauma was instrumental in the development of the lesions. Nikolsky's sign was strongly positive.

He returned two weeks later after using his hands as little as possible in the interim. Boric acid ointment U. S. P. was applied locally twice a day. His condition was definitely improved, although Nikolsky's sign was still strongly positive. He also presented several crusted lesions on the face, which were apparently the result of shaving. There was also some scaling of the ears.

During the past two weeks he has been taking 60,000 U. S. P. units of vitamin A and 50,000 U. S. P. units of vitamin D daily.

DISCUSSION

DR. HARRY E. ALDERSON: I questioned the patient in detail. It is interesting that the lesions have been strictly limited to his hands, and he apparently has never had any lesions on any other part of his body. He is well informed about the compensation laws, and I strongly suspect that the lesions might be self inflicted. Why do not lesions appear on the feet or buttocks?

DR. HIRAM E. MILLER: This eruption has been present for only eight or nine weeks, and he has had it before. I wonder whether he might have a prolonged erythema multiforme.

DR. ERVIN EPSTEIN, Oakland: He was doing gardening for the WPA when the lesions developed. He is not seeking compensation, but in another month he will be eligible for SRA relief. His face is also involved to a lesser extent. Careless shaving causes the formation of lesions on his face. Also, he has a strongly positive Nikolsky's sign. There is no erythema surrounding the bullae. The patient told me he had had no previous similar dermatosis.

It is interesting to realize what an economic handicap this condition is. Once while he was walking along the street somebody dropped some potatoes out of a bag. He picked some of them up, and lesions developed from even so little trauma.

A Case for Diagnosis (Plastic Induration of the Penis). Presented by Dr. E. A. Levin.

P. L., a white man aged 75, stated that in June 1939 he first noted a superficial pea-sized erythematous papule which broke down to form an ulceration on the glans. In a few weeks three or four more similar lesions developed. Some of them gradually healed, but for the most part they remained. There was no pain but some tenderness. As the process progressed, the patient noted the development of deep induration on the glans and distal portion of the shaft. Despite this, there was no particular urinary difficulty.

There was no history of gonorrhea, syphilis, trauma, insect bite or previous ulcerations over the penis. There had been no recent loss of weight. Tuberculous glands were removed from the neck fifty years ago. Wassermann reactions of the blood in June, August and September were negative.

Since there was the possibility that the process was a gumma, the patient was given five weekly injections of mapharsen, 0.04 Gm. each, the first given on August 25. This was followed by eight intramuscular injections of bismuth subsalicylate in oil, of 1 cc., given at weekly intervals.

The ulcerations have healed. The deep induration has remained stationary

DISCUSSION

Dr. George V. Kulchar: I do not believe that this condition is plastic induration of the penis. In respect to the possibility of syphilis, the patient apparently did have a gumma, and he responded rather rapidly to antisyphilitic therapy. Also, the shaft is not involved, as occurs in plastic induration.

Dr. N. N. Epstein: I believe that this man's induration does go well back to the shaft along the ventral surface almost to the base. The ulcers seemed to me to be syphilitic, but the induration may not be.

Dr. Hiram E. Miller: I do not believe that this condition is due to syphilis. The Wassermann reaction has been repeatedly negative, and the response to antisyphilitic treatment has been slight. The ulcers healed, it is true, but none of the induration has disappeared under antisyphilitic treatment.

Dr. H. J. Templeton, Oakland: Fuhs has successfully treated some patients with plastic induration of the penis by radium.

Dr. E. A. Levin: I do not know the exact diagnosis. The use of filtered roentgen ray therapy is worthy of trial.

Occupational Eczema Simulating Knuckle Pads (Heloderma Simplex Annulare). Presented by Dr. Arne E. Ingels.

G. S., a white woman aged 45, an artist (painter), presented herself Nov. 1, 1939, with a padlike thickening on the side of the third finger of her right hand. The "pad" is somewhat hyperkeratotic, infiltrated and loosely fixed to the underlying tissue. There is some evidence of irritation of the skin of the rest of the hands.

The patient has been bothered with this "pad" for some time, evidently several years, and has noticed that when she handles much turpentine and pigments in painting, it becomes pronounced, whereas it recedes or disappears at times when she is not active in her work. Being concerned about the lesion, she admits having manipulated it and also biting it, at times rather hard. The paint brush does not come in contact with this area. She has used only hydrous wool fat. No change in the lesion has ensued.

Because of the transient nature of the lesion, it is obviously an eczema due to mechanical and chemical irritation and not a knuckle pad.

DISCUSSION

Dr. Charles Albert Shumate: Dr. Ingels and I saw at the San Francisco City and County Hospital 2 patients with similar conditions, and both, on close questioning, said they had bitten the areas.

DR. GEORGE KULCHAR: In going over the histories of cases of heloderma in the literature, it is evident that many of the conditions are due to trauma; but there are others that are not. In the case I presented at the last meeting (ARCH. DERMAT. & SYPH. to be published) there were similar lesions on the feet. I am not prepared to state that trauma is not a factor in producing knuckle pads, although I do not believe it is the sole cause; I presuppose a nevoid background.

Allergy Due to Cold. Presented by DR. C. J. LUNSFORD, Oakland.

B. B., a girl aged 18, a student, about a year ago first noticed a burning and swelling of those parts of her body which were exposed to cold. This was true whether she held a glass of cold water in her hand, bathed or swam in cold water or exposed her face and hands to a cold wind. There is no history otherwise of allergy. She has no other complaints. The results of her general physical examination were negative except for a basal metabolic rate of — 17 per cent, for which she is taking thyroid.

When I first saw her I had her immerse her hands in cold water. After twenty minutes there was moderate but noticeable swelling.

During the last six weeks she has received twice a week ascending doses of 1 : 1,000 dilution of 1 per cent histamine phosphate, without noticeable benefit.

Atopic Dermatitis. Presented by DR. FRANCES TORREY.

E. J., a white woman aged 45, a telephone operator, applied for treatment of eruptions about the neck, shoulders and axillas which she had had for one year. When she was first seen, in September 1939, the areas were slightly erythematous, lichenified, fairly symmetric and pruritic. There was no history of childhood eczema, asthma or hay fever, although she is said to have had some eruption on her face and body. A maternal aunt had had eczema.

In 1934 the patient had had a similar eruption. A résumé of the observations made at that time, when she was seen by Drs. Templeton and Lunsford, is as follows: Routine cutaneous tests gave negative reactions except with orris root and onions. The patient improved with roentgen ray therapy and local treatment and was discharged as clinically cured in six weeks. She was treated in 1936 for contact and seborrheic dermatitis of the neck and in 1937 for alopecia areata and an erythematous vesicular eruption on the ear lobes.

Examination in the University of California Medical Clinic revealed nothing pertinent to her cutaneous complaint. She has used no cosmetics during the past year. Local applications have given symptomatic relief.

DISCUSSION

DR. H. J. TEMPLETON, Oakland: When we treated this patient about five years ago I regarded her eruption as a contact dermatitis, and I still think that it is, even though the offending agent has not yet been found.

DR. GEORGE V. KULCHAR: Has potassium chloride been tried?

DR. FRANCES TORREY: Not that I know of.

DR. MERLIN TREVOR-ROPER MAYNARD, San Jose: Contact with orris root cannot be eliminated by the patient simply changing the powder she uses. A woman cannot go into a room with other women present without having such contact. One patient I had went with his wife to a lodge meeting during which the women in their routine marched across the room in front of him, and he came into the office the next day with an acute recurrence.

DR. FRANCES TORREY: In this patient, however, the lesions are much more severe on the covered parts of the body, the axillas and the groins.

Acne Conglobata. Presented by Dr. Eugen Ostwald (by invitation).

A German-born white man, a lawyer aged 43, was first seen on Nov. 7, 1939. His past history was without significance. His father died of leukemia. There were no cutaneous diseases in his family.

At the age of from 20 to 25 the patient suffered from acne vulgaris on the face and neck and from "abscess" on his left arm.

The present condition appeared on the neck and trunk about ten years ago, beginning as deep pustules. Up to November 1938 he was treated almost continuously with chemical cauterization, sulfur baths, injections of a gold compound and sulfanilamide. The drugs had no effect.

During the past year his condition has been worse. The lesions have become painful and slightly pruritic and have caused great discomfort by the drainage of "pus" with a foul, penetrating odor. The patient always has been tired and has had a slight fever from time to time. No bromides or other drugs have been taken.

The patient has an oily skin. His face shows many varying-sized, white, depressed, sclerotic, irregular scars. On his neck there are many similar scarred areas, keloids, fibromatous partly pendulous scars, tunnels and bridge scars. Comedos and papulopustular lesions are scattered over his body. Some are large and occur in groups. There are papulofollicular lesions and elevated plaques of papillomatous and pustular type, with serpiginous and cicatricial development. A small amount of pus and decomposed sebaceous material exudes from these inflamed lesions and surrounds the patient with a penetrating foul fetor. The borders of the plaques are well defined. Occasionally communicating lesions can be seen, so that pressure on one will cause pus to appear from another nearby.

The surface of the involved areas is rough and irregular. There is a certain tendency for central healing. Some lesions are covered with small dark brown crusts and are reddish blue; here and there are thin-walled abscesses. The lesions are found particularly on the posterior and lower part of the body, but smaller lesions are on the front part also.

Serologic examination for syphilis gave negative results.

A blood count showed 4,050,000 erythrocytes and 13,800 leukocytes per cubic millimeter and 59 per cent hemoglobin, with a color index of 0.72; a differential count showed polymorphonuclear neutrophils 89 per cent, small lymphocytes 8 per cent; large mononuclear lymphocytes 2 per cent; eosinophils 1 per cent and basophils 0 per cent. The sedimentation rate was 25 mm. in one hour. Urinalysis gave negative results.

After roentgen ray treatment (250 r) the lesions appear to be less inflamed than when first seen. The patient has been given a diet rich in vitamins B_1 and C.

DISCUSSION

Dr. George V. Kulchar: I think the use of staphylococcus toxoid would be worth while in this case. I have seen a few conditions respond satisfactorily.

Dr. Harry E. Alderson: I wonder if any autoinoculation experiments were tried. The organisms might grow on his skin and not on another person's skin.

Dr. Eugen Ostwald (by invitation): I know that Michelson and Allen tried autoinoculation and transmission to other patients, but they succeeded only in autoinoculation. It seems that there might be a specific agent in the abscess lesions of acne conglobata that is not found in the lesions of ordinary acne, but this agent is not sufficient in itself to cause the specific lesions of acne conglobata if transmitted to other persons. Apparently another factor that is present only in the patient is necessary. This patient has a flabby skin with little turgor and seems to have little resistance.

Dr. Hiram E. Miller: I agree with the diagnosis. In this patient there certainly does not seem to be any reason to consider the condition to be a tuberculid. Dr. Maynard and I had a patient some years ago with a similar eruption and an associated iritis of questionable tuberculous origin.

DR. MERLIN TREVOR-ROPER MAYNARD, San Jose: The case Dr. Miller has just mentioned was one that I presented some years ago. I treated the patient ultimately with roentgen rays, with practically complete healing. One lesion that I treated with solid carbon dioxide failed to heal—why I cannot say. He continued to have his iritis and still has.

DR. EUGEN OSTWALD (by invitation): The von Pirquet test gave a slightly positive reaction in this patient. Other signs of tuberculosis could not be found. I first saw him ten days ago. At that time he exuded such a penetrating odor that it was impossible for other patients to enter the room where he was treated. He is much better now. I am giving him ascorbic acid (vitamin C), following a suggestion by Nicolau. During the World War Nicolau (Nicolau, S.: *Ann. de dermat. et. syph.* **7**:399, 1918-1919) observed a peculiar eruption, similar to acne conglobata, which he described as "follicular papulokeratotic scorbutic dermatitis." He was successful in treating these patients by giving them an antiscorbutic diet. I am trying the same with this patient because, as in Nicolau's patients, his condition is accompanied by anemia, general weakness and malaise.

CHICAGO DERMATOLOGICAL SOCIETY

EDWARD OLIVER, M.D., *President*

HERBERT RATTNER, M.D., *Secretary*

Nov. 15, 1939

A Case for Diagnosis (Histiocytoma?). Presented by DR. S. W. BECKER and DR. M. E. OBERMAYER.

E. T., a Mexican woman aged 40, was first seen in the Medical Clinic of the University of Chicago, complaining of swelling of the ankles, face and hands and shortness of breath of two years' duration. The symptoms were worse in summer. The history is inadequate because of her difficulty in understanding English. The patient had many miscarriages, and in 1922, after a positive Wassermann reaction was obtained, she was given some intravenous and intragluteal therapy. Further details about her illness could not be determined, aside from the fact that she had taken some pills for her heart.

The significant findings on physical examination were a slightly enlarged heart with a systolic murmur, palpable liver and edema of the hands and feet.

The Wassermann reaction was weakly positive and the Kahn reaction, negative. Examination of the urine showed a trace of albumin. A blood count showed 8,800 leukocytes and 5,570,000 erythrocytes per cubic millimeter; the differential count showed 73 per cent polymorphonuclear leukocytes, 14 per cent lymphocytes, 3 per cent mononuclear leukocytes, 8 per cent eosinophils and 1 per cent basophils. The basal metabolic rate on two occasions was + 37 per cent. The cholesterol content of the blood was 156 mg. per hundred cubic centimeters. No medical diagnosis was made.

Examination reveals moderately firm subcutaneous flesh-covered nodules from 0.5 to 1.5 cm. in diameter on the hands, arms, legs and thighs, which have been present for three to four months. These nodules are not tender and are both superficial and deep.

The patient has been seen on three occasions in the past two months, and the lesions seem to be regressing.

DISCUSSION

DR. M. R. CARO: I though that clinically and histologically the condition was not histiocytoma. In the cases Dr. Senear and I reported (ARCH. DERMAT. &

SYPH. **33**:209 [Feb.] 1936), the lesions were superficial and gave the impression of being inserted into the skin. In this case the lesions are situated at various depths. In our cases histologic examination showed that the infiltration extended up to the epidermis; in this case there is a thin layer of normal corium between the infiltrate and the epidermis. In our cases the infiltrate extended into the surrounding tissues; in this case it is well circumscribed.

DR. UDO J. WILE, Ann Arbor, Mich.: I was impressed by the difference between the clinical and the microscopic picture. There was considerably more cellular activity shown microscopically than was suggested by the clinical picture, and I was struck by the microscopic resemblance of the lesion to a rapidly growing sarcoma.

DR. S. W. BECKER: We realize, of course, that this is not the same type of tumor as the growths described by Drs. Senear and Caro. Those were individual tumors which were formerly called fibroxanthomas but are now usually called histiocytomas. We studied this infiltrate for a long time. Dr. Delaney, of the department of pathology, assured us that the cells of the tumors were histiocytes and not tumor cells. The peculiarity of the miscroscopic picture is that the infiltrate is perivascular and is not in one single location. The patient states, and I believe from our observations that we can corroborate her statement, that these lesions are receding. No primary focus of any tumor has been found. We are still not quite clear as to what the disease is.

Dermatitis with Depigmentation (Pellagra?). Presented by DR. E. P. ZEISLER.

A rapidly spreading eruption developed on the arm, backs of the hands, scalp, forehead, groins and ankles of a Negress aged 14 after an injury or bite on the forearm several weeks ago. She was seen in the clinic six weeks ago, and a diagnosis of impetigo was made and ammoniated mercury prescribed. After this treatment she rapidly became worse and was hospitalized five weeks ago. At that time she presented a sharply demarcated parchment-like dermatitis of the back of the hands, extending around to the wrist and on one arm above the elbow, with loss of pigment in the exposed areas. There were also oozing, eczematous lesions on the elbows, in the vulvar region and around the ankles. On the forehead was a white depigmented patch. In spite of the absence of gastrointestinal and oral lesions, the diagnosis of pellagra was considered because of the glovelike distribution of the lesions on the arms and the peculiar distribution of the eczematous patches. A blood count showed 13,500 leukocytes per cubic millimeter and secondary anemia.

The child was rather vague in answering questions about her diet, and there appeared to be mild mental symptoms associated with the dermatitis. After the oral administration of nicotinic acid and iron, soaking in potassium permanganate and the application of naftalan ointment, rapid recovery ensued. At the present time the depigmentation at the site of the eruption is the most conspicuous feature.

DISCUSSION

DR. S. W. BECKER: I believe the condition in this case could be classified as a leukomelanoderma on a postinflammatory basis. Since the pigment is now returning, perhaps most or all of the involved areas will regain their normal color.

DR. E. A. OLIVER: In seems to me that of late a great many cases of depigmentation have been observed. Dr. Tauber recently sent me a report of a case of depigmentation in a Negress who had worn rubber dress shields. She gave a definite reaction to a patch test and showed depigmentation in the axillas, which may have been the result of contact with agerite alba. This is the substance which was found to be the cause of depigmentation in Negroes who wore a particular brand of rubber gloves. In the case now under discussion I cannot see any evidence of pellagra.

DR. E. P. ZEISLER: I am told that depigmentation in Negroes is rather common, but I cannot say that extensive depigmentation following eczematoid dermatitis is common. The use of ammoniated mercury may have had something to do with it.

Regarding the diagnosis of pellagra, when I saw the patient originally it was merely a tentative diagnosis. There was no history of diarrhea or of oral lesions but merely of deficient food intake and anemia. As far as therapy is concerned, nicotinic acid had a decided healing effect.

A Case for Diagnosis (Oil Tumors?). Presented by DR. HERBERT RATTNER and DR. THEODORE CORNBLEET.

N. B., a Negress aged 50, has a history of addiction to morphine for the past twenty years. She gave herself eighteen to twenty injections daily, and now she maintains that she has not taken any morphine since 1938. About a year ago a nodule appeared on the outer surface of the left arm at the site of the injections. New lesions of a similar character then appeared adjacent to this. In the past few months nodules have appeared on the right arm. Now both arms are the sites of deep and superficial pea-sized nodules, situated in slate gray patches, the skin overlying some of the lesions being reddish and scaly. There are no subjective symptoms.

She received antisyphilitic treatment at times up to 1935, and in 1938 a colostomy was performed for rectal stricture due to lymphogranuloma venereum. The Kahn reaction was negative.

A roentgenogram of the chest revealed increase of hilar markings, with slight calcification in the hilar lymph nodes.

The blood picture was normal; the urinalysis showed albumin (2 plus).

Histologic examination showed a sarcoid-like structure, with multinucleated giant cells and numerous ovoid spaces.

DISCUSSION

DR. M. OPPENHEIM (by invitation): This is an interesting case. The microscopic observations suggest the diagnosis of sarcoid or lupoid. There were epithelial cells, giant cells and few round cells, all aggregated in sharply limited nodules. Not all patients who receive injections of morphine or other substances get such lesions. I do not know whether the presenters have found any foreign substance in these lesions. One must differentiate between paraffinoma (oil tumors) and sarcoid caused by foreign bodies. In paraffinoma one finds mostly giant and epithelioid cells surrounding the oil. From 1906 to 1912 I observed a series of cases of tumors starting from intramuscular and subcutaneous injections of various remedies. I considered these reactions as foreign body reactions. Later, when I knew more of sarcoid or lupoid, I came to believe that these lesions might be a typical reaction of certain persons, the so-called sarcoid reaction. An attenuated or a dead tubercle bacillus may cause such a sarcoid or lupoid reaction (Kyrle). The more virulent the tubercle bacilli, the more round cell infiltration is found. In cases of syphilis and leprosy the lupoid or sarcoid reaction is occasionally observed. I have found the development of sarcoids in syphilitic patients after intramuscular injections of mercuric salicylate and bismogenal. In 1 case it was seven years after the injection in the buttocks that sarcoid-like tumors appeared. Then I noticed such growths after injections of morphine, arsenic and iron, and after injections of soluble substances, when it was supposed that horn cells transported from the epidermis by the needle into the body caused the sarcoid reaction. Volk, of Vienna, made experiments with injections of china ink and could produce the lupoid reaction in certain persons. It seems to me that persons suffering from tuberculosis or syphilis are showing this kind of reaction more frequently. It is interesting that this patient who is presented to demonstrate sarcoid also has syphilis. At the Congress of the German Dermatological Society in Vienna, in 1912, I read a paper stating that one can have a sarcoid reaction to any kind of foreign body, whether an inorganic substance, like morphine, oil, horny scales or

cells or an organic substance, like bacteria. Van Studdiford (van Studdiford, M. T.: Diagnostic and Therapeutic Use of Tuberculin in Certain Acneform Eruptions, ARCH. DERMAT. & SYPH. **38**:737 [Nov.] 1938) stated the belief that lupoid or sarcoid tumors are due to tuberculosis with attenuated tubercle bacilli.

DR. HERBERT RATTNER: We thought it interesting that there were nodules on the inner surface, where presumably no injections had been given, but experienced observers assure me that the site is not an unusual one for such injections. I had assumed, because of the character of the lesions both clinically and histologically, that at some time oil had been used as the vehicle for the drugs injected and that the condition was oil tumors producing a so-called sarcoid-like histologic picture.

DR. THEODORE CORNBLEET: I thought that the foreign body reaction might be due to the fact that this woman had used hypodermically morphine tablets intended for oral use. Such tablets have some inert excipient mixed with the active ingredient to give bulk. The reaction could be due to an inert powder present.

A Case for Diagnosis (Sarcoid of Boeck?). Presented by DR. HERBERT RATTNER.

Mrs. H. K., a white woman aged 42, states that about one year ago a pimple appeared on the right side of the face near the mouth and another on the left temple, which persisted despite the use of various remedies. On squeezing one she obtained serosanguinous fluid. New papules appeared adjacent to the first ones; and these have formed reddish coin-sized patches of agminate papules and nodules, some of them traversed by telangiectases. The outline of the patches is somewhat polycyclic. A bartholinian abscess was removed one year ago. There is a history of two miscarriages interspersed with two normal pregnancies and no history of tuberculosis in the family. The Kahn reaction was negative.

The report on the histologic section was "sarcoid structure."

Sarcoid of Boeck. Presented by DR. D. V. OMENS and DR. M. H. EBERT.

M. B., a negress aged 40, first noticed a nodule on her left upper eyelid in the latter part of 1936, which slowly became larger. New nodules developed on other eyelids, around the alae nasi and mouth and on the cheeks. Some of the lesions have been removed because of obstruction to the nostrils. She has always been in good health. In 1936 a fibroid growth was removed from the right side of the spine, leaving a keloid at the site of excision. She also has several keloids on the arm and breast from old injuries. On the left breast is a pedunculated bluish warty mole. On the left thumb is an asymptomatic movable circumscribed round nodule under the skin. There is no history of tuberculosis in the family or contacts, so far as the patient knows. There is some enlargement of the anterior and posterior cervical glands. The patient has been pregnant three times but had three miscarriages at the third or fourth month of gestation.

Roentgenologic examination of the chest showed cardiac enlargement and an increase of hilar markings, with a slight infiltrative extension into the lung parenchyma. Roentgenologic examination of the hands showed some decreased density in the terminal phalanx of the third finger of the left hand and of the right thumb.

A Mantoux test with 0.1 cc. of a 1:100,000 dilution, gave a negative reaction. Examination of the ears, nose and throat revealed that the alae and vestibule of the nose were involved by reddish brown papules and nodules. The superior border of the epiglottis was thickened and clubbed. Examination of the eyes disclosed no pathologic changes.

Examination of the blood showed 67 per cent hemoglobin, 3,930,000 erythrocytes and 5,500 leukocytes per cubic millimeter, with a differential count of 68 per cent polymorphonuclear leukocytes, 1 per cent eosinophils, 1 per cent basophils, 26 per cent lymphocytes and 4 per cent monocytes; there were hypochromia (2 plus) and anisocytosis (1 plus).

DISCUSSION ON CASES OF DR. RATTNER AND DR. OMENS AND DR. EBERT

DR. UDO J. WILE, Ann Arbor, Mich.: I am in accord with the diagnosis of sarcoid in the case of the Negress who had multiple lesions on the face and eyelids. The condition in the other case, however, looked much more like a syphilid, and the histologic picture was not that of sarcoid, as I ordinarily understand the term. In a general way, the introduction of sarcoid has made for unfortunate confusion in dermatologic terminology. The majority of sarcoid lesions are those of noncaseating tuberculosis. I fell that Dr. Oppenheim's discussion involving such terms as sarcoid reaction and tuberculoid reaction adds somewhat to the confusion. A tuberculoid reaction can and is frequently caused by a foreign body and is in no way associated with true tuberculosis. A cumbersome terminology has added confusion rather than clarity to the picture of cutaneous tuberculosis.

DR. HOWARD J. PARKHURST, Toledo, Ohio: I have observed cases like that of the Negress with multiple lesions on the eyelids and lips. I thought that case might be considered as one of what Klauder described as "sarcoid-like lesions occurring in the Negro."

DR. M. R. CARO: In regard to the Negress, she does have syphilis. She was under my care at the Mandel Clinic of the Michael Reese Hospital for about six months. Under antisyphilitic treatment the lesions did not improve. Sections from the lesion on the nose and one from the forearm both showed the histologic picture of sarcoid.

DR. HERBERT RATTNER: The Negress referred to by Dr. Caro was presented here two years ago by Dr. Ginsburg and me (ARCH. DERMAT. & SYPH. **36**:921 [Oct.] 1937). At that time we pointed out as an interesting feature the nodules in the mucous membrane of the nose. I noted that there were more of them today.

The white woman is from the outpatient department of Cook County Hospital. We presented her to the students as having syphilis. The serologic reaction was negative. Later the report of the histologic examination was sarcoid. Incidentally, there is a history of two miscarriages, and a flare-up was noted after the administration of iodides. I think the case is a good example to illustrate Dr. Oppenheim's point in regard to sarcoid reaction.

DR. M. H. EBERT: The case of the Negress was presented by Dr. Omens as an instance of the type of condition that Dr. Nomland studied and described and that he called sarcoid. It is a hematogenous tuberculosis of the noncaseating variety of a peculiar type seen in the Negro race.

A Case for Diagnosis (Fixed Eruption?). Presented by DR. M. H. EBERT and DR. D. V. OMENS.

V. B., a 32 year old Negress, never had an eruption until two months ago, when several red pruritic spots developed on the left foot and in a short time became blisters. Lesions then appeared on the right arm, and later the same type of lesion developed on the trunk and extremities, remaining as pigmented flat lesions. She gives a history of taking as laxatives anything available, such as magnesium sulfate and castor oil and pills and tablets she gets from friends but the names of which she does not know.

When she attended the dermatologic clinic for the first time she was given 1 grain (0.065 Gm.) of phenolphthalein. In fifteen minutes a generalized pruritus developed which lasted about half an hour, with no appreciable change in the cutaneous lesions. While in the ward she received 1 grain (0.065 Gm.) of phenolphthalein and two hours later another grain. On examination several of the lesions were inflamed and surrounded by an area of hyperemia, which lasted several hours. She did not complain of much pruritus at this time.

The other findings are an umbilical hernia, a small fibromyomatous uterus and hydrosalpinx on the right side.

The Kahn reaction was negative. Examination of the blood showed 70 per cent hemoglobin, 3,200,000 erythrocytes and 5,200 leukocytes per cubic millimeter, with a differential count of 49 per cent polymorphonuclear leukocytes, 1 per cent eosinophils, 43 per cent lymphocytes and 7 per cent monocytes. The urinalysis gave negative results. A patch test with phenolphthalein gave a negative reaction after thirty-six hours.

DISCUSSION

DR. D. V. OMENS: The resident physician told me that he gave the patient some phenolphthalein today just before she was presented. There was a slight flare-up of the lesions, which quickly subsided.

DR. L. F. WEBER: A patch test would not be expected to indicate anything in this patient. These conditions do not represent epidermal sensitization.

DR. M. H. EBERT: We tried the patch test and also administration of the drug. Both gave negative reactions. We thought that the first reaction she had was to the phenolphthalein that was administered. Other substances besides phenolphthalein produce fixed eruptions, and they must be investigated before we can state positively what caused the condition. I think it is definitely a fixed eruption.

Pyoderma Gangraenosum. Presented by DR. M. H. EBERT.

About Sept. 20, 1939, P. A., a Negro aged 55, noticed some "blisters" on the dorsum of the right hand and over the region of the right patella and lower part of the thigh. He opened all the bullae with a sewing needle, with resulting ulceration and gangrene. The two lesions about the knee were at first discrete but later coalesced to form the long lesion seen. He has been having mild fever since admission. His reflexes were found to be normal. He had been drinking 1 to 2 pints (500 to 1,000 cc.) of whiskey daily prior to the onset of the present complaint.

The blood pressure was 182 systolic and 92 diastolic. The urine was normal. The Kahn reaction was doubtful, and the Wassermann reaction was 4 plus. Blood examination showed 56 per cent hemoglobin, 3,200,000 erythrocytes and 15,000 leukocytes per cubic millimeter, with a differential count of 76 per cent polymorphonuclear lekuocytes, 1 per cent basophils, 12 per cent lymphocytes and 12 per cent monocytes.

The patient has a large ulcer on the external surface of the left thigh, about 4 by 20 inches (10 by 51 cm.). The floor is granulating and bright red. The wall is serpiginous. There is little infiltration. In places the skin is slightly undermined. There is also a small crusted ulcer on the left leg and one on the left flank.

DISCUSSION

DR. UDO J. WILE, Ann Arbor, Mich.: This man had a severe hypertension and peripheral arteriosclerosis. I think all these lesions could be explained on the basis of continuous infarcts, particularly if one saw them develop. Those lesions on the left leg began as small to large areas of necrosis which gradually coalesced and formed one of the large ulcerous conditions. I think the lesions are cutaneous infarcts.

DR. LESTER M. WIEDER, Milwaukee: I got the impression of an advancing necrotizing process, such as occasionally occurs about the genitalia and in which the causation is often obscure. Two lesions on the medial aspect of the thigh were rupial, and I believe the possibility of an unusual syphilid must be considered. The location and type of many of the lesions suggest that other factors than senile degenerative vascular changes must play a causative role.

DR. S. ROTHMAN (by invitation), Paterson, N. J.: Even if an infarction is the primary cause of the lesion, an infection must be assumed, because the edge of the lesion is serpiginous in many places. In noninfected infarctions the edges are sharp and regular.

DR. UDO J. WILE, Ann Arbor, Mich.: I have not infrequent opportunities to see patients, mostly older persons, who are in poorhouses or county homes. For the most part, they are arteriosclerotic. Such patients are prone to have cerebral accidents, and not infrequently thromboses and emboli are apt to develop and result in infarcts in the skin. I should like to ask Dr. Ebert if his patient has had attacks of intermittent claudication and muscle cramps. These frequently precede the onset of infarcts, and acute infarction results in the development of a blue-black circumscribed plaque, which rapidly sloughs out.

DR. M. H. EBERT: I must apologize for the incomplete work-up of the case. The patient was seen for the first time two days ago, and nothing particular was done except the Wassermann test, which gave a positive reaction.

I thought of three possibilities that have been mentioned. My first impression was that the condition was a variety of pyoderma gangraenosum but it lacked some characteristic phases. Most of those conditions are in patients who have had ulcerative colitis and a considerable loss of blood, with severe anemia. This man did have secondary anemia, with the hemoglobin down to 56 per cent, but there was no history of colitis. However, the serpiginous border was present, and when I saw him on Monday I thought I saw one area where the gangrenous epidermis was undermined, and I felt there was progression at that point. There has been considerable treatment by surgeons. The area has been debrided and bathed with diluted solution of sodium hypochlorite U. S. P., so it is hard to say what it looked like at first, except the beginning lesion on the left leg. Thinking the matter over after leaving the ward, I felt that the most likely diagnosis was the one Dr. Wile suggested: necrosis on the basis of arteriosclerosis. One must remember that the man has syphilis, and it is possible that the gangrene which had started from some other cause might be influenced by the syphilis.

DR. D. V. OMENS: Dr. Ebert and I have seen a patient with a condition similar to this, an ulceration of the lower extremities due to arterial infarcts. At that time a woman, about 70 years of age, presented a discrete type of lesion on both legs below the knees. It was on the basis of an arteriovenous disturbance. In the present case the lesions are diffuse and coalesced, and it hardly seems that there is any resemblance between the 2 cases.

NOTE.—The lesions in this case healed rapidly after administration of anti-syphilitic treatment.

A Case for Diagnosis (Psoriasis Pustulosa?). Presented by DR. JAMES H. MITCHELL.

Mrs. W. J. C. was first seen on Nov. 11, 1939, with symmetric onychia and lesions on the palms and soles. She stated that she had been in a train wreck on Nov. 12, 1938, and that five hours later intense itching and an eruption had appeared on her palms and soles. There was also some mild itching of the arms. Later the nails became involved. She was seen by various physicians and spent some time in a sanitarium. The basal metabolic rate was — 30 per cent at the time of onset, but ingestion of thyroid substance had brought the rate up to — 15 per cent when last determined. There have never been any lesions in the scalp or on the elbows or knees. The general health, aside from nervousness due to itching, has been excellent. Dr. Cleveland J. White had the patient under observation for some months and had some roentgen ray treatments given. There was no evidence of tonsillitis or dental infection.

Clinically the lesions consist of symmetric, scarcely visible, superficial, minute vesicles containing clear watery fluid, with scattered pinhead-sized scales in the centers of the palms. The finger nails show pitting of the distal portion, typical of psoriasis. There are various changes about the free and lateral borders of the nails characteristic of psoriasis. No fungus could be found in the lesions of either

the skin or the nails. The vesicle content was sterile on brain broth medium. Insufficient time has elapsed for cultures to develop.

The lesions on the feet consisted of palm-sized, well defined areas of erythema with some lichenification and numerous minute superficial vesicles in various stages of involution and evolution and were located on the inner sides of the arches. A magnifying glass was necessary to detect the presence of the vesicles. The changes in the toe nails were similar to those of the finger nails. No fungus could be found either in the lesions on the arches or in the nail substance.

DISCUSSION

DR. FRANCIS E. SENEAR: There have been many cases of this type presented here in which there was a question as to whether the diagnosis was pustular psoriasis or acrodermatitis. This is the first condition I have seen in several years that I can accept as pustular psoriasis. The picture on the skin itself is no different from what has been seen in other cases, but from the changes in the nail plates and the itching the condition seems to be psoriasis.

DR. S. W. BECKER: Patients with similar lesions are seen and are studied a great deal. I have never been able to culture fungi from the pustules. The nails show pitting but seldom as much thickening as these nails show. Heller has called attention to the fact that pitting occurs in "eczema of the nails" as well as in psoriasis. Several years ago I presented a patient with nail changes (ARCH. DERMAT. & SYPH. **24**:698 [Oct.] 1931), who had had the typical picture that this patient presents along with dermatitis of the sides of the fingers. I interpret this condition as a functional dermatosis but realize that it is a bad name. I do not believe there is any way to clear up this condition except to institute treatment for functional disease. The onset following the train wreck is not surprising. These conditions often follow a sudden shock of that type.

DR. E. A. OLIVER: Do you believe that this eruption would develop five hours after a train wreck?

DR. S. W. BECKER: I have seen eruptions come on shortly after a shock.

DR. HERBERT RATTNER: This case has a striking resemblance to those reported from Belleview Hospital (Bloom, D.: ARCH. DERMAT. & SYPH. **32**:90 [July] 1935) a few years ago, in which the condition was histologically shown to be psoriasis.

DR. L. F. WEBER: I should not like to testify that the condition is a functional dermatosis following the train wreck.

DR. M. H. EBERT: I think this condition is the best illustration one could find of what Barber has termed pustular psoriasis. The nail changes are characteristic of psoriasis. The lesions on the palms and soles, their location, the character of the epidermal changes and the type of scaling are all as he described. Confusion arises in attempting to distinguish pustular psoriasis from recalcitrant eruptions of the palms and soles. In the latter there are no psoriatic plaques in other areas and no changes in the nails. I think Barber feels that the removal of a focus of infection is salutary and often results in a complete cure.

DR. E. A. OLIVER: I have had 2 of these cases that were interesting. In 1 a man had a severe recalcitrant eruption of the palms and soles, such as that described by Andrews, and showed characteristic psoriasis of the nails. The condition cleared up nicely after massage of the prostate gland and the removal of several abscessed teeth. I also saw a woman recently who had the same type of recalcitrant eruption. When I first saw her she told me that she had had thirty-five roentgen treatments on the palms and soles. I advised the removal of several abscessed teeth, and shortly after the removal of these foci she began to improve. She has been entirely well now for practically three months, and I am convinced that the removal of foci of infection is an important procedure.

DR. J. H. MITCHELL: I should like to ask Dr. Becker if he would be willing to go on the stand and say that this condition was the result of the wreck.

DR. S. W. BECKER: I would not say that the condition would not have developed without the wreck. Some of these eruptions develop gradually. Sometimes such a condition will develop after a severe shock. I would simply state that the wreck could have precipitated the eruption.

DR. FRANCIS E. SENEAR: On what ground do you call the eruption a functional disease?

DR. S. W. BECKER: I call this condition a functional disease on the basis of the studies that have been made. I have found that these patients all have abnormally active nervous systems. While I realize that any diagnosis of functional disease may be questioned, I have obtained excellent results in many patients treated for functional disease. The changes in the nails of this man cleared up promptly. He was shown later with juxta-articular nodes due to syphilis, at which time his nails were normal (ARCH. DERMAT. & SYPH. **31**:744 [May] 1935).

No one knows why a cutaneous disease will develop in one patient and colitis in another. Many patients have come with cutaneous disease which I call functional. They have at times been to a half-dozen clinics, and in each place diagnosis of functional disease, such as colitis or migraine, has been made, and they come to me with a functional cutaneous disorder. The basal metabolic rate in these patients has been universally low, — 15 per cent, which is a low normal. When Dr. Wilder was chairman of the department of medicine he said he did not know why these patients have low metabolic rates; they have low blood pressures as well.

DR. FRANCIS E. SENEAR: Did you say that you get uniformly good results?

DR. S. W. BECKER: The treatment goes back to the rest cure. The patient takes a daily nap and a daily sunshine treatment and is given crude coal tar ointment locally. Patients are also given what has been termed "home-made psychotherapy," simply common sense discussion of their problems. Functional disease is interpreted on an energy basis. As a psychiatrist said some weeks ago, he is perfectly able to discuss neuroses even with the physicist because he considers them on an energy basis. These patients have an energy deficiency without a normal sense of fatigue. They keep on overdoing. By forcing them to rest a feeling of fatigue appears, and the functional disease improves.

DR. E. A. OLIVER: Do you have a follow-up study of these cases?

DR. S. W. BECKER: I follow a few. I have been studying them for twelve years, and every once in a while a patient returns. They give a history of having almost complete freedom of the condition except at times of stress, which cause recurrence.

DR. J. H. MITCHELL: I have been thinking about this subject for many years, but I have never had a clear conception of what psoriasis pustulosa actually is. This comes nearer to being that condition than any I have seen. Going through the literature rapidly I found that sudden onset in the absence of anything like a train wreck is commonplace. Whether she would have had this without the train wreck is a different question. I had a feeling that these were characteristic psoriatic nails. The pitting is proximal rather than distal.

The functional treatment of these so-called functional conditions is not always a success, as many patients, after a year of phenobarbital and rest in bed, are still having trouble.

Dermatitis repens is not to be considered in this case. That is a staphylococcic infection with a high degree of virulence, and there is no response to treatment. It is usually preceded by injury. The difference between pustular bacterid and pustular psoriasis is difficult to decide, but pustular bacterids are not sterile. The lesions of pustular psoriasis, according to every one who has studied them, are sterile, and that happens to be the case here. The nails are characteristic of psoriasis.

For the information of Dr. Becker I should like to say that his assumption that patients are cured because they do not return to him may be at times erroneous. Comparing notes with my colleagues leads me to the conclusion that not a few of the patients supposedly cured have sought treatment elsewhere.

EDWARD A. OLIVER, M.D., *President*

HERBERT RATTNER, M.D., *Secretary*

Dec. 20, 1939

Condom Dermatitis. Presented by DR. DAVID V. OMENS.

M. L., a married salesman aged 48, presents an inflammatory dermatitis of eight months' duration involving the genitals and adjacent areas including the anal cleft. There have been periods of remissions and exacerbations of variable length; however, the exacerbations seem to occur on his return home from business trips. He has used rubber condoms for contraceptive purposes for many years, and, so far as he knows, always the same brand.

The urinalysis gave normal results. A patch test with a condom gave a positive result. (The patch test was demonstrated.)

DISCUSSION

DR. M. E. OBERMAYER: It might be worth while to treat the condoms with a 4 per cent solution of sodium hydroxide, as I suggested several years ago. In the manufacture of gloves and condoms, sulfur monochloride is often employed in the process of cold cure vulcanization, and if the hypersensitivity is due to this compound, eczema will be prevented by treating the material with alkali before it is used.

DR. HERBERT RATTNER: I reported such a case a few years ago, and it led to considerable correspondence with public health institutions in various places. My patient had in addition to dermatitis a nonspecific urethritis, which apparently was due to the rubber. And after the report, according to these letters, a number of similar cases were discovered, 17 cases from one institution alone. One social service group learned that the offending rubber in each instance was made by one firm. In my case, Dr. Obermayer's suggestion of alkalinizing the rubber proved effective.

DR. L. F. WEBER: I have recently in several instances had experience with rubber dermatitis from the wearing of rubber gloves. During the last few years rubber gloves have been worn more in some industries. I have also observed cases of rubber dermatitis from the wearing of rubber respirators. There was some material used in the manufacture of the respirator which irritated the skin about the mouth and nose. After new respirators were substituted the dermatitis disappeared. My experience with alkalinization of the rubber has been somewhat different from that of Dr. Rattner. I have not had occasion to check the manufacture of the rubber respirators, but I will, since they are made in Chicago.

DR. S. W. BECKER: One of the physicians in Boston told me he had seen depigmentation produced by the wearing of rubber dress shields.

Cheilitis Due to Deficiency of Riboflavin. Presented by DR. MAX S. WIEN and DR. ALBERT H. SLEPYAN (by invitation).

G. E., aged 34, first noted a scaling eruption on his lower lip in 1925. He thought it followed an abrasion from a cigaret on the vermilion portion of the lower lip. The abraded area improved slightly and then broke open again, with the formation of crusts at the injured site, until 1927. He is employed in a food packing plant and eats a general diet, with a moderate amount of meat and bread and a minimum amount of fresh vegetables. His general health otherwise has been good.

He was first seen by us in September 1939, at which time he had a heaped up, honey-colored crust involving the entire vermilion border of the lower lip. The crust was firm and adherent. After conservative local treatment, the crust was separated, and there were revealed an underlying dry scaling of the lower lip with

fissures and a clinical picture much like that presented today. The lip has been completely clear at times after treatment with vitamin B complex tablets or with capsules containing 1 mg. of riboflavin, or approximately 400 Sherman-Bourquin units of vitamin G (B_2). The prompt response to treatment with riboflavin prompted us to think that we were dealing with cheilitis due to deficiency of riboflavin. This idea was further strengthened after reviewing the work of W. H. Sebrell and R. E. Butler on "Riboflavin Deficiency in Man" (*Pub. Health Rep.* **53**:2282 [Dec. 30] 1938). These writers noticed that in 10 of the 18 women given a riboflavin-deficient diet there developed "a cheilosis (lesions on the lips) in from 94 to 130 days after the beginning of the experiment. These lesions began as a pallor of the mucosa of the lip in the angles of the mouth without involvement of the buccal mucosa. This pallor was soon followed by maceration, and within a few days superficial transverse fissures appeared, usually bilateral, and exactly in the angle of the mouth. . . . The lesions remained moist and became covered with a honey-colored crust which could be scraped off without bleeding."

Examination today reveals a crisp tissue-paper-like crusting of the lower lip, with occasional fissures where there is a loss of continuity in the crust. There is no evidence of hemorrhage or lesions on the buccal mucosa.

DISCUSSION

DR. HARRY R. FOERSTER, Milwaukee: Of interest in this case is the observation of beginning involution of the lesion and subsidence of inflammation following the use of the vitamin B complex. This would help to rule out consideration of both lupus erythematosus, which is a possible clinical diagnosis, and lichen planus, which is a less likely diagnosis.

DR. FRANCIS E. SENEAR: Dr. Wien called my attention to the fact that I had seen this patient in 1937. On looking up the record I found that Dr. Stillians had also seen the patient and presented him to the society with the diagnosis of lupus erythematosus. According to the patient's statement, when I saw him that diagnosis was not entirely accepted. However, if the man's history is to be relied on, I do not think conclusions should be drawn too hastily, because he said that the attacks last one to two weeks and have a spontaneous remission, and then he is free for one to two weeks. Dr. Stillians had treated him with many things. Just about the time I saw him Dr. Rattner spoke of a similar case in which the condition was cleared up with vitamin C, so I administered vitamin C, and he recovered while under that treatment. The last note I had in the history was that he had on the occasion of his last visit only two tiny points, which showed no crusting, but he said that the lip was beginning to feel thickened, which was always a premonitory sign that he would have another attack. Whether avitaminosis is the causative factor or not seems to me to be doubtful.

DR. CLARK W. FINNERUD: This is certainly an instructive case if the condition is due to a deficiency of riboflavin vitamin G (B_2). But I am still not wholly convinced that the disease is not lupus erythematosus.

DR. MAX S. WIEN: I first saw this man in September 1939, with the history of a crusted dermatitis of fourteen years' duration on the lower lip. Three days after he was given riboflavin the skin cleared. He presents none of the aspects of lupus erythematosus at this time. The lip is not tender. One lesion which still looks blue is a bit sore from manipulation before the presentation.

Subacute Disseminated Lupus Erythematosus. Presented by DR. ERWIN P. ZEISLER.

A symmetric erythema of the face developed in a woman, aged 23, after a sunburn in July, which has persisted to date and has recently appeared on the extensor surfaces of the arms. The eruption is dull red and superficial and occupies the flush areas of the cheeks, forehead and chin, with blotchy lesions on the upper extremities. There is a fever every afternoon with a temperature up to 101 F.

The blood count showed 80 per cent hemoglobin and 6,560 white blood cells per cubic millimeter, with a differential count of 54 per cent polymorphonuclear leukocytes, 32 per cent lymphocytes, 12 per cent monocytes and 2 per cent basophils. The Wassermann and Kahn reactions were negative. Treatment to date has consisted of local application of mild preparations. Iodoform and iron were given internally for ten days, without benefit.

Lupus Erythematosus with Acrosclerosis. Presented by DR. DAVID V. OMENS and DR. WALTER W. TOBIN (by invitation).

M. M., a woman aged 49, states that about seven years ago red patches began to appear on her nose, left temple and scalp, which became progressively larger and scaly and on healing left white scars. Six years ago she noted red areas on her finger tips and face, which have increased in number, especially during the last two years. The fingers, and during the last two years the toes as well, have at times

Roentgenogram of the hands of a patient with lupus erythematosus with acrosclerosis.

become purplish and numb and have felt "like needles and pins." The symptoms are much worse during cold weather, but the face seems to be worse during the summer.

Examination discloses smooth, atrophic, whitish scars on the left side of the nose, region of the left temple and in both ears and extensive involvement of the vertex of the scalp. On the scalp and in the ears there are areas of bright erythema, scaling and follicular plugging. There is a tightening of the skin over the cheeks and fingers, with numerous telangiectatic points, which disappear on pressure. The hands and to a much less extent the feet show sclerotic islands which are firmly attached and on removal leave a punched-out ulceration. There is considerable shrinkage of the fingers, especially of the right hand, producing a clawlike hand.

The results of serologic tests of the blood, a blood count and a urinalysis were normal. The basal metabolic rate was + 10 per cent, and tests with old tuberculin in dilutions of 1 to 100,000 and 1 to 1,000 gave negative results. A roentgenogram

of the chest was normal; however, roentgenograms of the hands showed destruction of the distal phalanges of all the fingers and thumbs of both hands, with small areas of calcification in the soft tissues over the ends of the fingers. Also there was some atrophy of the bones of the hands.

A histologic examination of an area on the scalp showed hyperkeratosis, thinning of the epidermis, a bandlike lymphocytic infiltrate of the corium and degenerate connective tissue. Histologic examination of a telangiectatic area on the neck showed little change except for dilatation of the blood vessels.

DISCUSSION

Dr. Hamilton Montgomery, Rochester, Minn.: I have observed 3 cases in which lupus erythematosus was associated with generalized scleroderma or acrosclerosis, without this fact being, I believe, of etiologic significance. It was just a coincidence, two diseases occurring simultaneously.

In Dr. Zeisler's case, I favor the diagnosis of disseminated lupus erythematosus. I did not see any typical keratotic plugs, but histologic studies would definitely determine the diagnosis.

Dr. Oliver S. Ormsby: Having just lost a patient with disseminated lupus erythematosus acutis, which developed on the subacute type, this case is of interest to me. I think subacute disseminated lupus erythematosus is not necessarily of the discoid type, although the discoid type may be related and develop into the acute disseminated variety. I have seen that happen a number of times. For example, 1 of my patients who had the common fixed discoid type suffered a severe sunburn, and a generalized multiform erythema-like group of symptoms immediately developed, which terminated fatally. The condition was in reality lupus erythematosus disseminatus acutis. The other patient, who recently died, had ordinary subacute disseminated lupus erythematosus for seven months. Six weeks ago this developed into the acute type, represented by acute constitutional symptoms, which included elevation of temperature and general toxemia. At autopsy no cardiac lesions were found, and there was no sign of tuberculosis. The condition was therefore not an example of the Libman-Sachs syndrome but apparently an infectious process, probably of streptococcic origin. The terminal conditions were acute meningoencephalitis and bronchopneumonia. Hemolytic streptococci were cultured from the blood. Generalized subacute disseminated lupus erythematosus without constitutional symptoms may continue for a long period, and the patient may recover, or it may develop into the acute type. In a number of these patients the cutaneous symptoms do not resemble those of ordinary lupus erythematosus. At autopsy in my cases endocarditis, pulmonary or glandular tuberculosis or a streptococcic infection have been the most frequent findings. In regard to the cases in which the face is involved, erythematoid lupus vulgaris (Leloir) is a combination of lupus erythematosus and lupus vulgaris. In reality it represents lupus vulgaris of the face which is unusually erythematosus. Lupus pernio of Hutchinson consists of an early transient erythema of the face, which eventually becomes permanent and is in reality discoid lupus erythematosus. The lupus pernio of Besnier is a form of sarcoid. In this form cystic osseous lesions are often present in addition to the lupus-erythematosus-like sarcoid lesion of the face. Lupus miliaris disseminatus faciei is a more acute type in which the lesions appear in crops and disappear spontaneously leaving pits. In these typical caseation is found. The rosacea-like tuberculid of the face (Lewandowsky) presents small bluish or reddish brown papules or papulopustules in the rosacea area. These conditions can usually be distinguished from other tuberculoses of the face. In the future all these conditions will probably be described as tuberculosis with some qualifying adjective to denote the type.

Dr. E. A. Oliver: In the autopsy in this last case were any renal changes found?

Dr. Oliver S. Ormsby: No.

DR. E. A. OLIVER: What do you consider the proper therapy for subacute lupus erythematosus?

DR. OLIVER S. ORMSBY: My best results have been obtained with a series of injections of sodium thiosulfate followed by a series of injections of a bismuth compound. I never like to give these patients gold preparations, because I have seen this condition change from the subacute to the acute type, terminating fatally. Before bismuth was introduced I used iodoform and iron.

DR. JAMES H. MITCHELL: I recently observed a case of typical lupus erythematosus in which the condition reacted much like this one on the face, symmetrically. The young woman had most extraordinary sensitivity to light. The smallest dose of rays from a quartz lamp would give a sharp reaction. It was not an example of the Libman-Sachs syndrome, so far as I could determine. She did not have the low leukocyte count associated with the acute type. Her general health was fairly good. I feared to give her a gold compound, because I felt the same way as Dr. Ormsby does. After lesions developed on the arms in August I decided to try small doses of quinine. She had two doses of 1 grain (0.065 Gm.) each, and a generalized, intense purpura developed and went on to a point where I thought she was going to die. She had four blood transfusions. Whether the quinine caused this or not I do not know. I shall hesitate in the future to try this form of medication. She is in good condition now but has a moderate amount of erythema on the arms and face. She has gained about 4 pounds (1.8 Kg.) in weight.

DR. M. H. EBERT: I should like to discuss the case presented by Drs. Omens and Tobin. Some years ago there was a case at Rush Medical College, University of Chicago, that I presented before this Society on two occasions, in which there was acrosclerosis similar to that in the case presented today except that it was more extensive in the former. In that case the terminal phalanges had been absorbed, and there were localized deposits of calcium on the backs of the hands and forearms. On examination this was found to be calcium carbonate. The same sort of deposits were seen today in the roentgenogram of this woman's fingers.

The patient presented today, like the one I described, has sclerosis of the face as well as of the hands. When I attempted today to draw down the eyelids I found it was impossible, because the skin is fixed. There was considerable tenseness over the face and some induration about the lesions themselves on the face.

I was inclined to think that the lesions on the face in this woman were dilatations of the peripheral vessels. A few months ago I presented a case (ARCH. DERMAT. & SYPH. **40**:830 [Nov.] 1939) of linear morphea on the extremity in which the new lesions consisted of dilated blood vessels. I have watched some of these, and they have become sclerodermatous. I think it is generally recognized that the original change in scleroderma is in the vessels. I suggest that the lesions on the face are not lupus erythematosus but another manifestation of scleroderma.

DR. S. W. BECKER: I thought the most interesting feature was the presence of macular telangiectatic plaques, which could be associated either with lupus erythematosus or scleroderma. I would place the condition in the group of generalized scleroderma rather than in that of acrosclerosis, because there was some thickening of the skin of the face and arms. At the meeting of the Mississippi Valley Dermatological Society in Minneapolis (ARCH. DERMAT. & SYPH. **35**:973 [May] 1937) a patient was shown with the same combination of symptoms.

DR. OLIVER S. ORMSBY: At the International Congress in Budapest, Selle showed a number of patients with acrosclerosis and distinguished this condition from scleroderma. In this country acrosclerosis of Selle has always been described as diffuse scleroderma. There are some observers who believe this type should be considered a separate entity.

DR. HAMILTON MONTGOMERY, Rochester, Minn.: I thought I saw typical keratotic plugs in the lesion on the cheek. Histologic examination should reveal a characteristic picture of lupus erythematosus, which is quite different from that of

acrosclerosis, although there may be vascular changes present in both. In regard to the treatment of disseminate lupus erythematosus, there is definitely danger of further dissemination of the disease from the use of gold sodium thiosulfate, but its judicious use often proves of value for the chronic eruptions and for a few of the subacute disseminate types. Treatment of the patient with disseminate lupus erythematosus should be individualized and should include rest in bed at the hospital and complete exclusion of sunlight, even that coming through the window pane. Usually after a period of rest and mild medication with quinine, a gold compound can be used cautiously. It is important to watch for increasing leukopenia, which is indicative of toxic reaction from the gold salts. Lupus erythematosus must be regarded as a systemic disease, even though gross or microscopic evidence cannot always be demonstrated at postmortem examination in the heart, kidneys and other organs. Of our patients with subacute disseminate lupus erythematosus, 47 per cent have died, but some of the rest soon recovered, with no more therapy than rest in bed and quinine, and some benefited by a combination of intravenous injections of a gold compound and roentgen ray therapy of the gland-bearing regions of the body, similar to the roentgen therapy used for lymphoblastoma.

DR. MAURICE OPPENHEIM (by invitation): I am surprised to observe so many cases of lupus erythematosus here. There are fewer cases of this disease in Vienna but more cases of lupus vulgaris. In 90 per cent of the cases of lupus erythematosus there are tuberculids.

In the first case, of a typical reddened condition of the face which spread to the arms, I should not make a diagnosis of lupus erythematosus, because some cases like this show the syndrome of poikilodermatomyositis of Petges and Cléjat. An article by Guy, Grauer and Jacob (Guy, W. H.; Grauer, R. C., and Jacob, F. M.: Poikilodermatomyositis, ARCH. DERMAT. & SYPH. **40**:867 [Dec.] 1939) suggested that a focal infection was the cause of poikilodermatomyositis. Poikilodermatomyositis can start as a form of lupus erythematosus. If one considers the lupus erythematosus of Libman and Sachs, one must think of a septic infection like Dr. Ormsby and the others described today. In the woman presented by Dr. Omens and Dr. Tobin, I should be careful in the diagnosis of lupus erythematosus. The fact that she has fever leads me to think that she has some septic infection as a cause of a beginning poikilodermatomyositis.

DR. ERWIN P. ZEISLER: I had one experience with a case of subacute disseminated lupus erythematosus a good many years ago when gold therapy was first used. The mistake I made was that I did not look at the patient's arms. He showed me the lesions on the face and neck. On the second visit I discovered lesions on the arms and elbows. By that time he was already beginning to show serious toxic effects from one injection of a gold preparation, a purpural stomatitis. He recovered. Since that time I have been careful not to use gold compounds in treating the disseminated type. Several patients have recovered with cautious bismuth therapy. I still think that the patient I presented has a subacute disseminated type of lupus erythematosus.

DR. DAVID V. OMENS: Dr. Montgomery said that the histologic picture of the lesion on the scalp was compatible with that of lupus erythematosus. The patient also had faint patches about the nose.

DR. WALTER W. TOBIN (by invitation): We used the term acrosclerosis because of the striking similarity of our case to those reported in the *British Journal of Dermatology* (**46**:523, 1934), wherein the vascular symptoms, the tightness of the skin, the telangiectasia and the punctate keratosis of both hands and the face were the outstanding characteristics. In scleroderma there are usually no preceding or associated vascular changes, and when the disease involves the hands it ordinarily involves the forearms first and then extends over the dorsa of the hands to the fingers.

Rosacea-Like Tuberculosis. Presented by DR. A. W. STILLIANS.

An American woman of German parentage, a telephone operator, aged 21, said that she had had the first evidence of pulmonary disease in the winter of 1937-

1938. In March 1938 she entered the City of Chicago Municipal Tuberculosis Sanatorium. At that time her sputum contained tubercle bacilli. By January of the next year her sputum was free of bacilli, and in the following month the eruption began on her face. It soon attained the extent now present and since then has persisted, becoming redder when she is warm or has rubbed her face and at other times without known cause, but it does not react to heavy meals or to nervousness, so far as she knows.

Examination of the urine and blood showed no abnormalities. Wassermann and Kahn reactions were negative. The Mantoux test with a 1 : 10,000 dilution of old tuberculin gave a positive result.

A healthy-looking, well nourished young woman presents an eruption of minute dull red to brownish red macules and maculopapules, 0.5 to 1 mm. in diameter, thickly but discretely arranged over the lower two thirds of the nose, on both cheeks to within a short distance from the ears, on the chin and in the center of the forehead. They are soft and under diascopic examination become light brown. At times they are on a red background; at other times the skin about them is nearly normal in color. Behind each ear, more so on the left side, there is a patch of diffuse dermatitis. The scalp shows little scaling. Under observation for about three months the eruption has not changed materially except as to the color of the background.

A histologic examination of a biopsy specimen from the right cheek showed thin epidermis without interpapillary pegs, with about five layers of prickle cells, no granular layer and a parakeratotic horny layer which was not thickened. In the upper part of the cutis there were a number of round or oval masses of infiltrate originating about the level of the superficial plexus of blood vessels and extending up to the basal layer and down, in the larger masses, into the upper part of the reticular layer. The infiltrate consisted of loose tissue, with many round cells, a fair number of epithelioid cells and a few plasma and giant cells. So far no tubercle bacilli have been found.

DISCUSSION

DR. CLARK W. FINNERUD: I feel that the condition should be considered an atypical example of rosacea-like tuberculosis. Some of the characteristic lesions of that disease, however, are lacking.

DR. M. H. EBERT: Dr. Wile called my attention to the fact that in the histologic section there was a typical tuberculous infiltrate, giant cells, lymphocytes and epithelioid cells, in the uppermost part of the corium, in fact compressing the epidermis. He felt, and I did too, that the eruption was a form of lupus vulgaris disseminatus faciei. I think the lesions on the face will come under that diagnosis, and possibly that is how the whole condition should be classified.

DR. HAMILTON MONTGOMERY, Rochester, Minn.: I agree with Dr. Stillians' diagnosis of rosacea-like tuberculid rather than tuberculosis cutis follicularis disseminatus. The latter disease shows considerable caseating necrosis histologically, whereas rosacea-like tuberculid shows a noncaseating sarcoid-like reaction, which is the type seen histologically in this case.

DR. O. H. FOERSTER, Milwaukee: It seems to me that the condition in this case differs in several essentials from the classic Lewandowsky type of tuberculosis. The lesions are smaller than those seen in the Lewandowsky type, and pustule formation is absent. This may be an example of the disseminated miliary type associated with rosacea.

DR. S. ROTHMAN (by invitation): The histologic features and the positive tuberculin reaction are more in favor of rosacea-like tuberculosis. In lupus miliaris disseminatus faciei usually a negative tuberculin reaction is found, as in sarcoid and lupus pernio ("positive anergic forms"). In my case the patient did not respond even to 10 mg. of old tuberculin. It is peculiar that just that form of cutaneous tuberculosis which shows the most typical tuberculous structure and the only one which regularly undergoes caseation is not tubeculin-sensitive and is resistant to any kind of therapy.

DR. MAURICE OPPENHEIM (by invitation): Many cases of lupus disseminatus follicularis are observed in Vienna. Kaposi described the first cases as acne telangiectodes. There is a difference between tuberculosis papulonecrotica and lupus follicularis disseminatus. In the case of Dr. Stillians nodules of nearly equal size are seen from that of a pinhead to that of hemp seeds. Rosacea-like tuberculid lesions are more different in type. Both conditions are often confused with acne rosacea. I believe the lesions in this case, which are so regularly disseminated on the cheeks and chin, with equal-sized nodules, give the picture of lupus follicularis disseminatus.

DR. A. W. STILLIANS: This eruption on the face appeared just a month after the first test showing that the sputum had ceased to contain tubercle bacilli. I was careful not to use the word Lewandowski, but I thought the term rosacea-like appropriate because of the distribution.

Lichen Nitidus. Presented by DR. MAURICE OPPENHEIM (by invitation).

A girl aged 7 years shows on the neck, on the flexor sides of both elbows and on the flexor sides of both knee joints groups of pinhead-sized flat-topped pale nodules without scales. The nodules are not coalescent, the color changes in some areas from yellow to light brown and the surface is waxy. The nodules are independent of the follicles, and in some areas form lines. There is no itching.

Histologically, the papules consisted of a sharply circumscribed collection mostly of epithelioid cells in the papillary layer, which causes an elevation of the hyperkeratotic epidermis. The Pirquet reaction was positive.

DISCUSSION

DR. OLIVER S. ORMSBY: I think the picture clinically and histologically agreed with the diagnosis of lichen nitidus.

DR. MAURICE OPPENHEIM (by invitation): It is unusual to see lichen nitidus in such a young girl. The condition is believed to belong to the tuberculids. Although this girl has a mildly positive Pirquet reaction, I do not believe the condition is related to tuberculid. The case is interesting with regard to the location of the lesions.

Melanoblastoma. Presented by DR. M. H. EBERT.

M. P., a white woman aged 66, states that she has noticed a bluish nodule on the inner surface of the left foot for the past ten years. Within the past nine months this nodule has grown, and new ones have appeared on the foot and leg. In the past few months the old lesion has ulcerated and become raised above the surface of the skin, so that the patient has been unable to wear a shoe. She was admitted to the hospital because of hypertrophic arthritis.

Examination shows ankylosis of the finger joints, elbows and knees. There is a golf ball-sized granulomatous mass on the medial side of the left foot that bleeds easily; it is surrounded by somewhat raised hard bluish nodules. There are various-sized satellite lesions distributed on the foot and leg. The left foot and leg are larger than the right. No regional adenopathy is palpable.

Examination of the blood showed 82 per cent hemoglobin, 3,900,000 erythrocytes and 10,600 leukocytes per cubic millimeter, with a differential count of 70 per cent polymorphonuclear leukocytes, 6 per cent eosinophils, 22 per cent lymphocytes and 2 per cent monocytes.

The results of urinalysis were negative. The melanin test gave a positive result. The Kahn reaction of the blood was negative.

Roentgenograms showed marked osteoarthritis in several of the intertarsal articulations of the left foot. There was no definite evidence of metastases. There was marked osteoporosis. A roentgenogram of the pelvis showed sclerotic changes about the head of the left femur and the acetabulum. Arthritic changes were

present at the sacroiliac joint. The heart was enlarged, and there was calcification in the aortic arch.

Histologic examination showed that the epidermis was somewhat acanthotic. The upper third of the corium was occupied by a cellular infiltrate separated into lobules by connective tissue septums. These lobules contained cells of varying size, with deeply staining nuclei and foamy, poorly defined cytoplasm. A few contained pigment granules. In places the rete pegs were continuous with the tumor cells. Below the tumor cells there was a zone made up of lymphocytes, histiocytes packed with pigment granules and a few plasma cells. A third zone occupied the lower level of the corium and was made up of lymphocytes and fibroblasts.

DISCUSSION

DR. JAMES H. MITCHELL: I should like to report on 1 patient who had this type of lesion on the outer side of the foot. She was presented here about two years ago. At that time I thought I had managed to control the condition with rather intensive doses of roentgen rays. The pigment disappeared, and the lesion was in fine condition. Later I found the lesion recurring and gave her intensive filtered roentgen rays, without much effect. She was seen by Dr. Ormsby in consultation later. Metastases developed in the groin and in the cranium, and she died.

DR. S. W. BECKER: Some of these conditions will metastasize slowly. Dr. Valade, of Detroit, sent me a histologic section of a palm, from which I made a diagnosis of lentigo maligna. The lesion was completely excised, and the patient had no trouble for five years. Then enlarged lymph nodes developed in the axillas, which showed nonpigmented melanoma. There was no local recurrence in the palm.

DR. THEODORE CORNBLEET: I have noticed in several patients who had deeply pigmented malignant melanomas that the urine showed no increased amount of pigment. Some of the metastatic lesions contain less pigment than the original one. On the other hand, the urine may show more evidence of pigment than the lesions. It seems therefore that the urinary pigment does not always reflect the degree of pigmentation of the cutaneous lesions.

DR. M. H. EBERT: I had planned to show 2 patients with malignant melanoma, but the patient with the more instructive condition of the 2 was sent home. He was a young man, aged 21, who had a brown, smooth, shiny mole on the right arm. This was injured playing football two years ago, and he had it excised. He had no trouble until a few weeks ago, when he complained of severe pain in the back. He was brought to the hospital, where it was found that the scar showed no sign of recurrence, but there was an apple-sized firm gland in the axilla, and he had numerous metastases over the body. I have sections showing the type of cell, which is melanoma. He also had melanin in the urine in large quantities. In that man's case there was a period of two years after the excision before clinical symptoms developed. Roentgenograms of the chest showed the lungs riddled with metastases, though there were no pulmonary symptoms.

In the patient shown today the growth is more slowly growing and more benign. The histologic picture I think is interesting, because in some of the sections there is a definite connection between the tumor cells and the proliferative rete pegs, which are markedly acanthotic in places. One can also see evidence of the defense mechanism that was set up, i. e., the zone of lymphocytes, connective tissue cells and reticulocytes below the tumor cells.

Carcinoma of the Palate and Alveolar Process. Presented by DR. F. J. KENDRICK (by invitation), Gary, Ind.

W. D., aged 64, has a sausage-shaped spongy, friable mass lying along the left upper alveolar process, about the size of the thumb and involving part of the hard and the soft palate. A roentgenogram shows involvement of the maxillary antrum.

The point of interest is that the patient states that up to two weeks ago he was wearing an upper dental plate, without discomfort. Further, according to his history, some sort of small growth was removed by cautery in the region of the present growth about a year ago.

Cutis Hyperelastica. Presented by DR. THEODORE CORNBLEET and DR. D. M. COHEN (by invitation).

V. T., a white girl aged 18, presents cutaneous lesions on both knees and elbows of sixteen years' duration. There are no subjective symptoms.

The individual lesions consist of round reddish-purplish sharply demarcated atrophic scars, with a velvety texture. The skin can be picked up and stretched from the underlying tissues for a distance of 5 to 8 cm. There is a hyperextensibility of the joints of the fingers and thumb. There are no hematomas or pseudotumors. The condition has been present since infancy. No other members of the family have a similar disorder.

Histologic examination showed the uninvolved skin to have normal elastic tissue; the elastic tissue of the involved areas was fragmented and frayed.

DISCUSSION

DR. HAMILTON MONTGOMERY, Rochester, Minn.: It is interesting to note with elastic tissue stain that the elastic tissue fibers are not swollen or degenerated but are clumped together about the blood vessels and dermal appendages, and then there is a clear space or gap in the connective tissue where no elastic fibers are to be found. The term elastic fibers is a misnomer in that the elastic fibers tend to bind down the tissue but are not as elastic as the collagen fibers. The irregular arrangement of the elastic fibers in this case, with areas in the cutis where there are no elastic fibers, might therefore explain the ease with which the skin can be picked up and stretched and its lack of adherence to underlying subcutaneous tissues.

DR. THEODORE CORNBLEET: We purposely removed some normal tissue for elastic tissue staining but could not detect any changes from the normal. There were, however, changes in the abnormal parts. The tissue did not show the redundancy of elastic tissue that we expected to find. I think it was Dr. Foerster who raised the question as to whether in these patients the elastic tissue changes could be found in the eyegrounds. I was told by an ophthalmologist that he could not find any changes in the elastic tissue in the eyes. This patient did not show the transverse linear scarring near the joints of the extremities that I have seen in some of the other patients with this disorder. I had thought that that was one of the diagnostic features of this syndrome.

A Case for Diagnosis (Lichen Planus of the Eyelids?). Presented by DR. S. W. BECKER and DR. M. E. OBERMAYER.

L. N., a married white woman aged 20, was seen at the University of Chicago Clinics on Nov. 15, 1939, complaining of gradual discoloration and pruritus of the eyelids of five months' duration. She had exposed herself to the sun a great deal during the summer. There was no history of drug intake except for a tonic containing an iron preparation.

The physical examination, including examination of the eyes by ophthalmologists, gave completely negative results. The blood picture was normal. The Wassermann and Kahn reactions of the blood were negative.

The skin of the upper and lower eyelids showed an eruption which surrounded each eye in the form of a band about 1 cm. in width, sharply limited toward the surrounding skin. The surface was brownish and reddish and showed fine adherent scaling. In places the skin appeared slightly atrophic, but we were not certain whether this was actually the case.

Lupus erythematosus, lichen planus and dermatitis medicamentosa, were considered in the differential diagnosis in the order named. A specimen for microscopic examination was removed, and it seemed to us that the slide showed the features of lichen planus rather than those of lupus erythematodes.

DISCUSSION

DR. JAMES H. MITCHELL: I am much in doubt as to the diagnosis either from the clinical appearance or from the microscopic section.

I have observed a number of cases of this kind in which there were typical lesions elsewhere. I have photographs of lichen planus lesions of the lids, but they do not resemble the lesions in this case.

DR. FRANCIS E. SENEAR: I did not see the microscopic section, but from a clinical standpoint I felt that lichen planus was a tenable diagnosis. Eruption of the eyelids in lichen planus has been reported more frequently in recent years. I am a little prejudiced in this case, because twelve or fifteen years ago Dr. Wien and I presented a case before this society and everybody insisted it was a case of lupus erythematosus because lichen planus did not occur about the eyes. The condition in that case was proved histologically to our satisfaction to be lichen planus. In the last couple of years we have had our attention called to lichen planus in this location; so it does occur with sufficient frequency to make the diagnosis probable.

DR. CLARK W. FINNERUD: From the gross appearance I expected to find lichen planus on histologic examination. The section shows lupus erythematosus, however, so I favor this diagnosis.

DR. HAMILTON MONTGOMERY, Rochester, Minn.: It may be difficult at times to distinguish histologically between subacute disseminate lupus erythematosus and lichen planus, but not between discoid lupus erythematosus and lichen planus. The section in this case is typical for lupus erythematosus. I have recently observed several cases in which the lupus erythematosus was confined almost entirely to the eyelids. This type of lesion might be amenable to gold therapy.

DR. M. E. OBERMAYER: From a clinical standpoint the condition appears more like lichen planus; however, on the basis of microscopic examination the diagnosis leans toward that of lupus erythematosus. As to Dr. Montgomery's suggestions, the leukocyte count appeared to be normal.

Bullous Disease. Bullous Darier's Disease (Pels and Goodman). Familial Benign Pemphigus (Hailey and Hailey). Herpetiform Dermatitis Repens (Ayers and Anderson). Presented by DR. S. W. BECKER and DR. M. E. OERMAYER.

E. McK., a housewife aged 40, complained of a chronic recurrent eruption in the axillas and on the sides of the neck of twenty-two years' duration. The eruption began as a vesicular erythematous patch on the back of her neck. There is usually some itching associated with the onset of the lesions. The lesions spread peripherally in the form of arciform and circinate pustular bullae with serous crusts and terminate after four to eight weeks. They always reappear in approximately the same location and are worse in summer.

Many of the patient's relatives have a similar eruption (27 of 78 in three generations) located in the same places. One male relative also had involvement of the groins. The eruption appears for the first time usually between the ages of 16 and 18.

The results of a general examination were negative. The blood pressure was 110 systolic and 80 diastolic. Examination of the skin showed on the nape of the neck and in the axillas several irregularly shaped erythematous exudative lesions with serous crusts at the periphery. Later examination showed some bullae along the margin of the original eruption on the neck. A pseudo Nikolsky sign over the adjacent skin could be elicited, consisting of denudation of small areas only.

The white blood cell count was 11,700 per cubic millimeter, and the red blood cell count was 4,700,000 per cubic millimeter. The differential count, bleeding time and clotting time were normal. The urine was normal. Porphyrin was not found in the urine. Two cultures from the lesions showed hemolytic Staphylococcus aureus. A patch test with potassium iodide gave a negative reaction. (A microscopic section was presented.)

DISCUSSION

DR. OLIVER S. ORMSBY: At the last meeting of the American Academy of Dermatology and Syphilology (Nov. 6-8, 1939) I had papers on bullous diseases on my program. I had seen the reports of Ayers and Anderson (ARCH. DERMAT. & SYPH. **40**:402 [Sept.] 1939) and of Hailey and Hailey (ARCH. DERMAT. & SYPH. **39**:679 [April] 1939) and believed that this was an entity not described before. The familial involvement is quite extraordinary in these groups. In Philadelphia Dr. Hailey brought a number of sections and clinical pictures, which were presented. It was agreed that histologically this entity could be a form of pemphigus. The clinical picture resembles a herpetic eruption, though Nikolsky's sign was present. From the evidence at hand I felt that these physicians were justified in classing the condition as a benign form of pemphigus. I recognize that pemphigus is usually fatal, but pemphigus erythematodes is one benign form. Hailey and Hailey reported a large familial group but not as extensive as that presented today. I think it is interesting that there have been three distinct reports all appearing within a year, and now Drs. Becker and Obermayer confirm these reports with a much larger group of cases than previously observed. The title familial benign pemphigus suggested by Hailey and Hailey appears the most descriptive of those presented. I think Drs. Becker and Obermayer are to be congratulated on their excellent work and presentation.

DR. THEODORE CORNBLEET: The second woman without lesions showed a definite Nikolsky sign on the neck when light friction was applied there.

DR. HAMILTON MONTGOMERY, Rochester, Minn.: I studied the sections in Drs. Hailey's cases and thought the histologic changes suggested pemphigus more than any other condition, and the sections definitely failed to show the features of Darier's disease. *Corps* and *grains* and *Lucken*, or spaces characteristic of Darier's disease, are shown in the slide from the case reported by Dr. Becker today, and thus both types of histologic change may be seen in these cases, which together with the clinical history and familial incidence make this condition distinct from either ordinary pemphigus or Darier's disease.

DR. S. W. BECKER: This subject is one of the most interesting that I have had a chance to study. The members of this family have been around Chicago for fifty years. They are of French-Canadian descent and have fairly large families. They had seen no dermatologist because they had accepted the fact that this condition was in the family and that it might or might not develop. If they did get it, they bore with it as best they could. In French they call this condition *dattes*, which probably is derived from the French term *dartres* for vesicular eruption. They may have confused it with herpes simplex. In the entire family there have been no members with characteristic lesions of Darier's disease, in fact, with papular lesions of any type; so I think Darier's disease can be eliminated.

When we first saw this patient the lesion looked like circinate impetigo. The lesions of 2 of these patients yielded cultures of Staphylococcus aureus. We made inoculation experiments in 1 patient on the normal and scarified skin but were never able to produce an impetiginous lesion. These patients have had many children, and in none of them has impetigo ever developed, while the mothers had active lesions. Hailey and Hailey cultured streptococci from their patient. The condition has not spread to any other part of the body. However, the dominant heredity is identical with that in cases of Darier's disease. We have 12 males

and 15 females, which is about the same percentage as among patients with Darier's disease, a percentage of 16 to 18, which is the same as we found in cases of familial and of acquired epidermolysis bullosa which we are now studying. The regions involved are characteristic. I do not believe from the observations we have made in these cases that this condition could be called pemphigus except that pemphigus means a bulla. We felt that we had tentatively ruled out dermatitis herpetiformis by obtaining a negative reaction to potassium iodide ointment.

Dr. Montgomery called attention to the dyskeratotic changes typical for Darier's disease. It seems to me that rather than utilize the names that have already been suggested, it would be better to give the condition a name which designates the process, such as "dyskeratosis bullosa hereditaria." This makes no effort at etiologic classification.

A Case of Ulceroglandular Tularemia. Presented by Dr. M. H. Ebert.

Sarcoid of Boeck. Presented by Dr. Herbert Rattner and Dr. Irene Neuhauser (by invitation).

E. M., a Negress aged 29, states that lesions began to develop about the alae nasi and eyelids five years ago and have gradually spread to the lips and scalp. There were no subjective symptoms. Last winter hoarseness developed which improved during the summer and became much worse with the approach of winter. She was admitted to the Cook County Hospital in October with sore throat, hoarseness and difficulty in breathing which was of three weeks' duration and was becoming progressively worse.

Direct and indirect laryngoscopic examination revealed slight injection of the structures of the larynx, marked subglottic edema and slight edema of the true cords. There was marked crusting of the subglottic space, almost completely occluding the subglottic chink.

She was later transferred to the dermatologic service for treatment of the cutaneous condition, which consisted of multiple firm, some smooth, some slightly scaling, reddish nodules and plaques with raised irregular borders located about the eyelids and nares and in the scalp.

Roentgen examination of the chest showed infiltrative changes in the lungs extending out from the hilar area. There were pathologic changes in the bones of the hands.

Wassermann and Kahn reactions were negative, and the urine was normal. The Mantoux test with a dilution of 1:10,000 showed a dime-sized area of erythema and a slightly palpable papule after forty-eight hours.

A biopsy of a lesion taken from the ala nasi revealed a marked acanthosis of the rete pegs, and in the papillary layer of the dermis, extending down to the reticular layer, were isolated granulomatous lesions composed of epithelial and giant cells. The biopsy report confirmed the clinical diagnosis of sarcoid of Boeck.

Psoriatic Erythrodermia. Presented by Dr. M. H. Ebert.

J. C., a white man aged 42, presents a generalized scaling erythroderma. He gives a history of dry scaly patches which appeared on the scalp and face twenty years ago. The elbows, knees and legs were progressively involved during the next few years. He noticed no seasonal variation. There were some remissions. Three weeks ago, while taking violent exercise, he felt feverish and noted the appearance of a fine red rash on his forearms. He went to bed, and in the next three days the eruption became generalized. The palms and soles were the last parts to become involved. The skin began to scale; at first the scale was fine, and later, coarse and flaky. For the past five months he has applied a 20 per cent ammoniated mercury ointment.

The patient appears ill and has a low grade fever. There is a universal redness and exfoliation, with considerable edema of the lower extremities. His red

blood cell count was approximately 4,000,000 per cubic millimeter, and his white blood cell count, 10,300 per cubic millimeter. The differential count was normal. Kahn and Wassermann reactions of the blood serum were negative. The results of chemical examination of the blood, including determinations of total lipids and cholesterol, were within normal limits.

Psoriatic Erythroderma. Presented by DR. M. H. EBERT.

F. G., a Greek aged 34, first noticed dry scaling patches on the sternum and between the scapulas in 1935. Gradually the extensors of the elbows and knees became involved, as well as the forearms. There was some improvement each summer. One month ago he commenced to take a tonic prescribed by his local druggist, the composition of which is unknown. A week later he noticed an exacerbation of his cutaneous lesions. This was followed in two days by generalized redness and scaling.

He now presents a universal erythroderma with large flaky scales. The palms and soles are spared. His temperature is normal. The red blood cell count was 4,000,000 per cubic millimeter, and the white blood cell count, 8,900 per cubic millimeter; the differential count was within normal limits; the Kahn and Wassermann reactions of the blood serum were negative. The results of chemical examination of the blood, including determinations of cholesterol and lipids, were within normal limits.

DISCUSSION

DR. HAMILTON MONTGOMERY, Rochester, Minn.: In cases of exfoliative dermatitis the histologic changes usually remain characteristic of the original dermatosis. It is important to take a specimen for biopsy from a region where there has been no local or roentgen therapy. I believe these cases are of psoriatic erythroderma and that the patients should respond promptly to the use of crude coal tar and ultraviolet irradiation.

DR. E. A. OLIVER: At the Veterans Administrative Facility the best results have been with the use of crude coal tar ointment and the alpine sun lamp.

DR. S. ROTHMAN (by invitation): Patients with psoriatic erythroderma have marked disturbances in their water metabolism. Instead of the normal 25 per cent, 70 to 75 per cent of the total water output is due to insensible perspiration, and correspondingly less water leaves the body with urine and feces. In spite of their extremely "dry" skin the water metabolism of those patients is similar to that of intensively perspiring persons. Apparently this is the explanation for the chilly feeling of patients with erythroderma. Also single psoriatic patches show increased perspiration, 200 to 300 per cent more than the surrounding normal skin. Hyperemia has not such an effect. As the process of keratinization is connected with loss of water, it has been assumed that in cases of psoriasis the increased perspiration is due to the overproduction of keratin.

DR. THEODORE CORNBLEET: I wonder whether that increase in perspiration is not connected with the fact that there is no visible sweating over psoriatic lesions. I have come to this conclusion after attempting on numerous occasions to induce sweating in them.

DR. M. H. EBERT: These 2 examples of psoriatic erythroderma were so similar that I thought they were worth showing. There is a rare type of psoriasis that shows a tendency to become erythroderma without any cause. I remember a patient Dr. Ormsby had in the Presbyterian Hospital in whom erythroderma developed on several occasions. The patients shown today have no such history. One was interesting in that he showed acute psoriasis. Dr. Ormsby has frequently pointed out that the treatment of psoriasis during the acute stage is different from that of the chronic stage. This patient had acute psoriasis, and ammoniated mercury ointment was applied. I think this treatment had a great deal to do with the initiation of the erythroderma. In the case of the other patient we are not sure of the precipitating cause.

CLEVELAND DERMATOLOGICAL SOCIETY

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Nov. 30, 1939

Multiple Epitheliomas; Ulceration Following the Use of a "Cancer Paste." Presented by Dr. C. G. LaRocco.

J. E. H., a healthy, vigorous man aged 77, has an ulcer on his back of about eighteen years' duration. His past history has no bearing on the present condition. He believes that his maternal grandmother died of cancer of the nose. Twenty years ago he had a carbuncle in the right scapular area. This was treated surgically, healed and left a circular scar with a radiating keloid. Because of his work as a planer in a wood mill he has had sores on his back frequently from splinters of wood. He has treated these himself. About eighteen years ago a sore developed in the midline of the interscapular region which has persisted.

Early in August 1939 a black "cancer paste" was applied to this area by a "cancer specialist," after which the area of ulceration increased.

There is a roughly oval ulcer deeply excavated, 8 by 5 cm., having a serosanguinous base and a piled-up, rolled and indurated border. Above this lesion and to the left is a tumor mass about 1.5 cm. in diameter, showing a pearly, rolled, indurated border with a crusted center. There are scattered keratoses over his back, with crusts and beginning ulceration in some of them.

DISCUSSION

Dr. John A. Gammel: I think we all agree with the diagnosis, but I should like to know about treatment. I had a case like this some time ago, and it was approximately a year before the site of the lesion was epithelized.

Dr. C. G. LaRocco: This patient was presented primarily from the standpoint of discussion as to treatment, and also I thought it might be of interest to the younger physicians to see what happens when this so-called cancer paste is used. I wondered about the treatment also and had thought that I might infiltrate the border and desiccate it, following this with roentgen ray treatment.

Dr. J. Edgar Fisher: This man is 77 years of age; I suggest treating the condition palliatively.

Dr. James R. Driver: This lesion clinically resembles the type of malignant growth known as Marjolin's ulcer. That is, it looks like the malignant type which sometimes develops in old varicose ulcers and in old scars, usually from severe burns. I think the appearance is due to the type of treatment he has had. In this type of case there is always a certain amount of cancer tissue locked up in scar tissue. Certainly there is plenty of scar tissue here as the result of the trauma of the cancer paste. It will therefore be more or less resistant to ordinary methods of treatment, except extensive destruction. However, I do not think I would treat the condition by extensive electrodesiccation, for it would be slow in healing. The case is ideal for interstitial irradiation, with radium needles of low intensity.

Dr. C. G. LaRocco: I have just returned from the Philadelphia meeting of the Academy of Dermatology and Syphilology, and while there I attended some lectures on roentgen ray and radium therapy. Dr. Hayes Martin, of the Memorial Hospital, who talked about cases like this, suggested roentgen ray treatments rather than radium therapy.

DR. EARL W. NETHERTON: In Pusey's textbook (Pusey, W. A.: The Principles and Practice of Dermatology, ed. 4, New York, D. Appleton and Company, 1917, p. 1054) there is almost an exact duplicate of the lesion in this case, although that lesion is larger. Some time ago I had the opportunity of seeing that patient; the lesions had spread and involved the spinous process, and the vertebrae were exposed. The lesion was much larger than when the picture was taken. The patient had had roentgen therapy and various types of treatment, but the condition had continued to spread. I believe the lesion in the case just presented will be resistant to treatment and will have to be treated energetically.

DR. H. N. COLE: I agree with the remarks of Dr. Driver. One has here a condition in which there are cancer cells running in between strands of scar tissue, and to attempt to treat this with roentgen ray therapy would be difficult, for the area is rather large, and one would have to be careful lest some of the tissue break down and never heal. I would be hesitant about using roentgen rays. I do think that with interstitial irradiation one could get a good result, since action on the cancer cells would be almost selective in its character. I observed a young man who had a large Marjolin ulcer on his knee secondary to extensive trauma many years previously. I used interstitial irradiation six or seven years ago in treating him, and now he is living and entirely well.

DR. H. A. HAYNES JR., Akron, Ohio: I should like to ask the opinion of the members regarding adequate surgical excision followed by skin grafting and closure.

DR. L. J. CARSON: I think that is a good idea. I have had cases of this type. I recall one woman I treated for eight months, without any healing. With skin grafting the condition healed in several weeks.

A Case for Diagnosis (Rosacea-Like Tuberculid of Lewandowsky?).
Presented by DR. JAMES E. ENGELER (by invitation).

J. M., a white man aged 49, presented from the Department of Dermatology and Syphilology, Cleveland City Hospital, states that the lesions on his face have been present for approximately thirty years. The eruption appeared first on the nose, later spreading to involve the cheeks. There have been periods of regression but never to complete remission. He has consumed large quantities of alcoholic liquors daily for the past thirty years. There has been no treatment either locally or internally.

The skin of the nose and adjacent areas of the cheeks is erythematous, and scattered over the surface are telangiectases, papules and pustules. No apple jelly tubercles were visible on diascopic examination.

Roentgenograms of the chest revealed a mottling in the upper part of both lung fields, with cavity formation in several areas.

Histologic examination of tissue removed from the left cheek revealed a thinned, keratotic layer, with moderate acanthosis and intercellular edema. Deep in the corium were several localized areas of a dense cellular infiltrate composed of numerous plasma cells, many epithelioid cells, small round cells, a few polymorphenuclear neutrophilic leukocytes and an occasional eosinophil.

DISCUSSION

DR. I. L. SCHONBERG: I should like to offer a diagnosis of rosacea, in view of the fact that the patient has had this erythema of the nose for a good many years and also since there is little scarring. There are a few suggestive lesions on the face but not the type found in rosacea-like tuberculid of Lewandowsky. In addition, there is lack of erythema on the sides of the face, as is often seen in this condition.

DR. HAL E. FREEMAN: This man has been a drinker over a number of years, which may explain the telangiectasia. The outbreak of the eruption is connected

closely with the beginning of his tuberculosis. It appeared shortly before he found he had pulmonary involvement and has continued unabated since then, which made me think it may have some connection with that condition.

DR. JOHN A. GAMMEL: There is no reason clinically why this case should not be simply one of rosacea, and I think the histologic picture is not against it.

DR. GEORGE W. BINKLEY: My diagnosis in this case was also rosacea. The duration of something like twenty-nine years and the clinical picture, with the erythema, telangiectasia, pinhead-sized pustules and enlargement of the nose, are classic. The results of histologic examination are consistent with those of rosacea.

DR. J. EDGAR FISHER: It was my impression this condition was rosacea.

DR. H. N. COLE: I concur with the diagnosis of rosacea, although the patient has pulmonary tuberculosis.

Purpura Due to Ingestion of Cinchophen. Presented by DR. GERARD DE OREO.

W. D., a white man aged 55, was admitted to the Department of Dermatology and Syphilology, Cleveland City Hospital, on Nov. 18, 1939, complaining of a rash that had been present for two or three days. For the past month he had complained of pains in the joints. On one occasion he was given a tablet of cinchophen, which afforded him prompt relief. After the ingestion of this he was aware of no cutaneous trouble. A week before admission he took another tablet of cinchophen (7½ grains [0.48 Gm.]) and three days later noticed that his skin was "broken out."

The patient is a poorly nourished though not acutely ill man. The right wrist and left knee are moderately painful on motion, and there is mild edema of the surrounding tissue. The mucous membranes and fundi are normal. There is no evidence of hepatic disease.

Over the lower part of the abdomen and more profusely over both legs are numerous purpuric lesions.

The blood urea nitrogen, icteric index, serum vitamin C and results of urinalysis were normal. Serologic tests for syphilis gave negative results. The hemogram was normal. The Rumple-Leede sign was negative.

After the lesions had completely disappeared a test dose of cinchophen was given, and the purpura reappeared.

DISCUSSION

DR. H. N. COLE: It seems to me that this case in a way is a good one in which to consider the possibility of Henoch's purpura. The patient has had arthritis and also has complained of pain in the abdomen. However, he has not had bloody stools. One would have to consider this in differential diagnosis. However, the facts that he does not have any evidence of hemorrhage into the mucous membranes of the alimentary tract and each time he has had cinchophen he has had a reaction make me think that the condition is a dermatitis medicamentosa rather than Henoch's purpura.

Dermatitis Repens. Presented by DR. H. N. COLE and DR. JAMES R. DRIVER.

J. E. L., a white woman aged 59, whose general health had been good, in March 1936 pricked the tip of her left middle finger with a sewing needle. The wound apparently healed, but about two weeks later a small pustule appeared in this area. This healed within a few days, but there were recurrences about once a month for the next six months. A roentgenogram at this time showed no osseous disease.

Pustules continued to develop more frequently, and in July 1938 another roentgenogram showed a moderate amount of osteoporosis on the lateral side of the terminal phalanx and one small area suggestive of osteomyelitis. After this an operation was performed, and the diseased portion of the bone was removed. The

wound healed, and in March 1939 a roentgenogram showed no evidence of osseous disease. However, pustules continued to develop at the tip of the finger.

Repeated Wassermann reactions were negative, but the Kline precipitation test gave a slightly positive result. Cultures showed Staphylococcus aureus and Staphylococcus albus. A hemogram and the results of urinalysis were normal.

She was given a course of ten injections of a bismuth compound and ten injections of mapharsen, but this had no effect on the lesion. The following additional therapy has been given: ten intramuscular injections of lactigen (a proprietary lipoprotein preparation of milk), several roentgen ray treatments, ultraviolet irradiation, the application of gentian violet, Castellani's carbolfuchsin compound and various ointments containing ammoniated mercury, tar, bismuth tribromphenate, salicylic acid, carotene and phenylmercuric nitrate, zinc peroxide dressings according to the method of Meleney and ten injections of an autogenous vaccine. None of this therapy has been of any value.

On the tip and sides of the terminal phalanx of the left middle finger the skin is erythematous and covered by multiple, superficial pustules, which undermine the upper layers of the epidermis. Crusting results from drying and rupture of the pustules. When the crusts are removed, there is revealed a raw, glazed, red surface. New pustules then form within a few hours.

DISCUSSION

DR. I. L. SCHONBERG: I suggest the administration of sulfanilamide in addition to the local application of 20 per cent ammoniated mercury ointment. I have recently had a case of pustular undermining dermatitis of the hands which showed many staphylococci and streptococci. Under sulfanilamide therapy this condition cleared. However, it recurred after the administration of sulfanilamide was discontinued.

DR. H. N. COLE: We presented this patient because of the rarity of the condition, for we thought some of the younger physicians would like the opportunity of seeing it. The condition seems to be characteristic as to the history and the type of local lesions with undermined edges, from which it seems to be spreading with pustules over the surface of the lesion. The disease has been resistant to every form of therapy we have tried.

NOTE.—Since presentation before the society the patient has been hospitalized. For twelve days sulfanilamide was administered, the blood level being kept between 9 and 11.4 mg. per hundred cubic centimeters. An abscessed tooth has been extracted. She was last seen on May 11, 1940. The finger has remained healed for two months, and no evidence of the disease remains.

Syphilis (Mucocutaneous Relapse or Reinfection?). Presented by DR. JAMES E. ENGELER (by invitation).

M. V. P., a white woman aged 33, in October 1929 was seen in the outpatient Department of the Cleveland City Hospital. Diagnoses of early syphilis, secondary stage, and pregnancy were made. She presented a generalized eruption, and the Wassermann reaction of the blood was strongly positive. Treatment was continued somewhat irregularly over two years. During this time she received a course of ten injections of old arsphenamine, three courses of ten injections each of neoarsphenamine, two courses of mercury inunctions and one course of ten injections of bismuth subsalicylate.

In October 1931 a spinal puncture was performed, and examination of the spinal fluid revealed a strongly positive Wassermann reaction. The gum mastic curve was of the dementia paralytica type. At this time the patient was hospitalized, and therapeutic malaria produced. She had ten paroxysms. Subsequently three courses of bismuth subsalicylate were given, with rest periods between each course of ten injections.

She was seen in May 1939, at which time the Kline reaction of the blood was negative.

About November 1 she presented herself at the outpatient department, complaining of an eruption on the palms, on the dorsa of the hands, on the arms and in the mouth. She had been divorced in March 1939 and stated that she had had no sexual intercourse since that time.

The eruption on the palms consists of reddish brown, scaling papules. On the arms and hands the lesions are maculopapular, with a few pustular lesions on the dorsa of the hands and fingers. On the mucous membrane of the lower lip there are characteristic mucous patches.

The Wassermann and Kline reactions of the blood were strongly positive. Dark field examinations of tissue from lesions on the palms and lip revealed Spirochaeta pallida. Aside from a 2 plus reaction in the 1 cc. dilution the Wassermann reaction of the spinal fluid was negative. The cell count, globulin and gum mastic curve were normal.

Histologic examination of the tissue removed from the palms showed hyperkeratosis, moderate parakeratosis and acanthosis, as evidenced by narrowed but elongated rete pegs. Diffusely distributed, especially in the middle and upper portions of the corium, was a heavy cellular infiltrate composed of numerous plasma cells, small round cells, epithelioid cells and occasional polymorphonuclear leukocytes. The small round cell and plasma cellular infiltrate was primarily perivascularly arranged.

DISCUSSION

DR. JOHN E. RAUSCHKOLB: I have been able to follow this patient since 1929, when she came to the clinic with a secondary eruption and pregnancy. She was treated, and later a lumbar puncture was performed, which revealed early syphilis of the central nervous system. She was given a course of malaria treatments and received more treatment subsequently, amounting to seven or eight years of good antisyphilitic therapy. Last spring her serologic reaction was negative, and she had taken more or less long periods of vacation between infrequent alternating therapy.

About a month ago she came in with typical, scaly, circular, pea-sized lesions of the palms. I remembered the patient and also knew the amount of therapy she had received, and syphilis seemed improbable. But at the same time she had a lesion on the lip which was clinically a typical secondary lesion. She was hospitalized and dark field examinations of tissue from the lesions on the palm and lips revealed S. pallida. Today the lesions have changed to a papulosquamous type, and other lesions have developed on the arms, feet and legs, which look more or less like a late secondary or an early tertiary type. Most relapses of this type come within four years after the infection. This patient has had what I should have considered adequate antisyphilitic therapy from the initial visit in 1929 to 1936. It is most unusual to see a mucocutaneous relapse, but I am in favor of this diagnosis.

DR. JAMES R. DRIVER: I am in favor of mucocutaneous relapse as the diagnosis.

DR. HAL E. FREEMAN: This condition obviously is tertiary syphilis if it isn't a reinfection, and there isn't any such diagnosis, according to the Cooperative Clinical Group's classification.

DR. JAMES R. DRIVER: I think the classification has exceptions.

DR. H. N. COLE: Here is a woman with lesions on the palms in which one finds spirochetes, which is unusual with early secondary syphilis. One might find the organisms of syphilis on the mucous membranes, but in the cutaneous lesions it would mean that the patient has poor resistance and perhaps malignant syphilis. However, I do not think this patient has malignant syphilis. I am rather inclined to classify the condition as a reinfection. It is an interesting patient for study.

DR. GEORGE W. BINKLEY: I believe it is agreed that clinically and morphologically the patient shows evidence of early syphilis, but it is not known whether this is reinfection from a residual focus of infection she might have had or, on the other hand, a new infection. Closer questioning may reveal recent exposures.

Lupus Vulgaris of the Face. Tuberculosis of the Buccal Mucous Membranes, Pharynx and Larynx. Pulmonary Tuberculosis. Presented by DR. HAL E. FREEMAN.

A. T., a Negro aged 37, presented from the Department of Dermatology and Syphilology, Cleveland City Hospital, gave a history of having had trouble with his skin since 1930. The condition began on his neck and forehead and has continued in the same locations despite his own local treatment with various salves. No relevant family history was ascertainable. There has been almost total deafness since about Oct. 1, 1939.

There is a discharge from both auditory canals. About the nares and the upper lip there is a crusted, erythematous eruption. In the cervical region there are multiple draining sinuses from old scrofuloderma. The uvula and most of the soft palate have been destroyed by a granulomatous eruption. On the midforehead there is a circularly arranged group of papules and small nodules, which are firm, shiny and erythematous. In the center of this ring the skin is partially depigmented and is atrophic in appearance.

The Gaffky index on admission in October 1939 was negative and now is II. The Kline reaction of the blood was negative. Serial roentgenograms of the lungs have shown the progression of a fine mottling in the upper half of each lung. The hemogram was normal.

Histologic examination of tissue removed from the involved areas showed thinning of the epidermis, with complete obliteration of the papillae. Throughout the corium there were numerous areas of caseation and numerous giant cells of the Langhans type. These areas of caseation were surrounded by small round cells, epithelioid cells and occasional polymorphonuclear cells. Typical pathologic tubercles were seen.

Tuberculoma of the Palate and Right Angle of the Mouth. Miliary Pulmonary Tuberculosis. Presented by DR. HAL E. FREEMAN.

A. P., a white man aged 32, presented from the Department of Dermatology and Syphilology, Cleveland City Hospital, has had a cough for seven months and a "sore mouth" for eight weeks. He has lost 30 pounds (13.6 Kg.) in weight in the past three months.

At the right angle of the mouth there is a proliferative, crusted and fissured lesion about 1 by 2 cm. in area. At the junction of the hard and soft palates there is a large, oval, pale, granulomatous mass, which is rather soft, somewhat friable and very vascular.

The hemogram was normal. The Kline reaction of the blood was negative. Roentgenograms of the lungs in October 1939 showed miliary pulmonary tuberculosis.

Histologic examination of tissue removed from the lesion on the palate revealed only a small bit of the mucous membrane epidermis in the available slides, but this appeared normal. The corium was diffusely infiltrated with small, round and epithelioid cells, and there were occasional plasma cells. There were many areas of caseation surrounded by small round cells, and epithelioid cells and occasional giant cells of the Langhans type were seen.

Treatment has consisted of five roentgen ray treatments, 100 r weekly, to the intraoral lesion and four like treatments to the lesion at the angle of the mouth.

DISCUSSION ON PAPERS OF DR. FREEMAN

DR. JOHN RAUSCHKOLB: I had the occasion some years ago to study 20 cases of tuberculosis of the mouth. In 18 of them there were tubercle bacilli in the sputum. Morrow, Miller and Taussig had previously published a paper stating that the sputum contained bacilli in nearly all the cases. I believe that in nearly all cases with oral involvement the condition is secondary to an active, fulminating type of tuberculosis. Nearly all such cases are fatal.

DR. H. N. COLE: In the second case I would not be surprised if tubercle bacilli were not found locally, or at least not easily, because this condition is the type that is spoken of as tuberculosis gummosa.

The first patient was interesting because the throat presented the picture that is often seen in connection with old tertiary syphilis with scar formation. However, close examination of the skin shows that there is a collection of eight or ten small papular lesions on the tip of the nose, and the lesion on the forehead is typical lupus vulgaris.

NEW YORK ACADEMY OF MEDICINE, SECTION OF DERMATOLOGY AND SYPHILIS

E. W. ABRAMOWITZ, M.D., *Chairman*

LEWIS B. ROBINSON, M.D., *Secretary*

Dec. 5, 1939

Erythroderma Psoriaticum with Tumor Growths (Epitheliomatous Changes). Presented by DR. OSCAR L. LEVIN.

L. G., a woman aged 37, is presented in collaboration with Dr. Howard T. Behrman, from the Mount Sinai Hospital. The patient has had universal psoriasis since 1919. She has been under my observation for twenty years. In 1922 a nodular eruption appeared on both legs and has been present ever since (Levin, O. L.: Universal Psoriasis Complicated by Tumor-Like Formations, ARCH. DERMAT. & SYPH. **12**:360 [Sept.] 1925). The nodules have increased in size since 1937, and several of them have ulcerated and have been subsequently excised.

The cutaneous lesions consist of a universal eruption in which the skin is livid red, lichenified and covered with yellowish white fine and heavy scales. A slight degree of diffuse alopecia is present, but there are no large areas free of hair except for the absence of eyebrows. The nails show pitting and hyperkeratosis. On the legs there are numerous pea-sized to walnut-sized, oval, nodular masses, some pale white and others pink. Several of these are superficially ulcerated and covered with a yellowish crust. A few smaller nodules are present on the thighs, and a quarter dollar-sized ulcerated nodule is present on the right buttock.

Repeated urinalyses gave negative results. Blood counts were normal except for 3 to 6 per cent eosinophils. Chemical examination of the blood showed normal dextrose, nonprotein nitrogen, cholesterol and esters, calcium, phosphorus and phosphates. The Wassermann and Kline tests gave negative reactions. The results of basal metabolic tests have never been below + 35 per cent. Cutaneous temperature readings have been high over the entire body. Roentgenograms of the chest and gastrointestinal tract were normal. Sigmoidoscopic examination in November 1938 showed the presence of the follicular type of melanosis coli.

In 1934 histologic examination of nodules showed an ulcerated surface and wild downgrowth of epidermal tissue in irregular strands. Mitosis and pearl formation were pronounced. The diagnosis of epithelioma was made. In 1939 a section showed acanthosis, parakeratosis and papulomatosis, with formation of verrucous-like structures. Papillary bodies as well as the upper part of the cutis were characterized by an unusually large number of dilated capillaries. The diagnosis was psoriasis verrucosa.

In addition to innumerable topical remedies, the patient has received autohemotherapy, arsenotherapy, hyperthermia and roentgen therapy, with but slight response. The autohemotherapy caused a moderate remission with subsidence of pruritus on several occasions.

DISCUSSION

DR. SAMUEL M. PECK: I think the results of the biopsies should be discussed in relation to each other. Although only two were reported in the history, at least two more have been made that I know of. This is not a case of exfoliative dermatitis. Every biopsy showed a typical picture of psoriasis. I do recall a section made in 1935 which strongly suggested an epithelioma of the squamous cell type. Another section of a nodular lesion showed a fibroma in the cutis with typical psoriasis over it. The latest biopsy showed a verrucous change superimposed on the psoriasis. I doubt whether this patient ever had a true epithelioma. Certainly with that diagnosis, further developments of this tendency should be seen by this time.

DR. ISADORE ROSEN: I have had many opportunities to observe this patient during the past eight or nine years. She presents an unusual clinical picture. I see many patients with various forms of psoriasis in clinic and hospital work, but I do not remember ever having seen one with such an extensive eruption which persisted for so long a time. She has a high basal metabolic rate, ranging from 40 to 50 per cent above normal. This is a common occurrence in patients with generalized erythroderma. Another clinical feature is that she never perspires. At one time fever therapy was tried, but her discomfort was so great on account of lack of perspiration that after three or four attempts the method was discontinued. Several years ago, while she was under treatment in the hospital, there were large areas on the trunk which were normal in appearance, but the tumor-like lesions on the lower extremities persisted. One of them ulcerated and after removal was reported to be a prickle cell epithelioma.

DR. DAVID L. SATENSTEIN (by invitation): I do not accept the diagnosis of an erythematous form of psoriasis in this case. I should be more inclined to say that the patient has an exfoliative dermatitis now, probably secondary to the psoriasis. In regard to the lesions on the lower extremities, as a result of the long-continued inflammatory process, the connective tissue has become hypertrophied. There is a good deal of hypertrophy with the verrucous type of psoriasis. I see this condition occasionally. Those who are not acquainted with it are apt to call it a malignant process, but it is not. It is a pseudomalignant condition. Years ago I used to encounter a number of these cases at the New York City Hospital. I consider that the condition on the upper part of the body is an exfoliative dermatitis and that on the lower portion a verrucous type of psoriasis.

DR. PAUL GROSS: The nodular and verrucous lesions in this patient were present only on the lower extremities, which points to another contributing factor. I saw a patient many years ago with typical psoriasis and large vegetating lesions on the legs, resembling bromoderma. In that case, varicose veins were apparently responsible for the unusual manifestation of psoriasis. The patient presented tonight shows no varicosities. Still there is a possibility of circulatory disturbance of the legs, clinically not recognizable. Dr. Rosen pointed out the high basal metabolic rate of this patient, which probably is due to the extensive involvement of the skin. On the other hand, it would be worth while to study the tumor-like lesions from the point of view of localized myxedema.

DR. GEORGE C. ANDREWS: Arsenic sometimes induces hyperplastic changes in the skin of patients who have psoriasis, and cancer on the legs may be caused by roentgen ray treatment. Roentgen therapy of the legs is probably fraught with more danger than that of any other part of the body, because of overlapping when the four surfaces are exposed. I have seen carcinoma occurring in a patch of psoriasis in only 1 case. Although psoriasis is frequently designated a precancerosis, it is unusual for a carcinoma to occur in a psoriatic patch. In the case I mentioned it occurred on the shin. It was an ulcerated basal cell epithelioma in a patch of psoriasis on the lower portion of the leg.

DR. HERMAN GOODMAN: The patient appeared to have had arsenic rather than psoriasis as the primary cause of the erythroderma, and the tumors, which might

easily have come from overdoses of arsenic or repeated small doses of arsenic, produce the picture of hyperkeratoses and epitheliomatous changes.

DR. OSCAR L. LEVIN: This patient does not suffer from a secondary dermatitis exfoliativa. When one sees the patient for the first time, the diagnosis of dermatitis exfoliativa is usually made. The cutaneous eruption is that of psoriasis and the pathologic changes are those of psoriasis and not of exfoliative dermatitis. When I first saw the patient in 1919, she presented a universal redness with scaling, and here and there the characteristic lesions of papular psoriasis were found. In regard to the action of arsenic, in my paper published in 1925 it was shown that there was no evidence of arsenic administered clinically, and the examination of the urine did not reveal the presence of arsenic. It was positively stated that the eruption bore no relation to arsenotherapy. Tissue which had been removed from an ulcerating lesion in 1935 revealed the presence of mitosis in numerous cells and the development of epitheliomatous pearls. The pathologist believed that the particular lesion which he examined was an epithelioma. However, no further changes of this kind have been observed since that time. The high basal metabolic rate is due to the fact that the universal erythroderma is associated with a tremendous loss of heat and the body works at a greater pace.

Mycosis Fungoides. Presented by DR. ISADORE ROSEN.

Y. C., a woman aged 60, is presented from the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital with a generalized eruption which began eight years ago. At that time a diagnosis of psoriasis was made. The eruption was treated with diachylon ointment topically for approximately four years. All therapy was discontinued three years ago. She has received ultraviolet irradiation and began the use of hormotone-T (Reed-Carnerick). She took two vials in several months. Six months ago she was given a series of injections of autogenous blood. About five months ago the patient noted that the eruption became crusted and new crusted lesions appeared at the sites of the injections of blood. The condition has since then slowly assumed its present appearance. She has refused all medical therapy since the eruption changed its appearance.

There is generalized lymphadenopathy. The liver is palpable 3 fingerbreadths below the costal margin, the border being sharp and the consistency firm but not hard. Pain is noted only on firm pressure. The spleen is not palpable.

Examination shows a generalized, discrete and confluent, polymorphous eruption. Early lesions are papulosquamous with thick scales, not easily removed, and brownish. Older lesions are infiltrated with erythematous borders. There are confluent plaques with ulcerations, in semilunar and annular configurations. Many are covered by heaped-up bloody crusts which are adherent. Several areas on the body show annular pigmented atrophic changes, apparently where old lesions have healed. There is diffuse blotchy pigmentation of the skin of the entire body.

Urinalysis gave normal results. A blood count showed 4,190,000 erythrocytes per cubic millimeter, a color index of 0.804 and 10,200 leukocytes per cubic millimeter, with 69 per cent polymorphonuclear neutrophils, 21 per cent lymphocytes, 4 per cent monocytes and 6 per cent eosinophils. Histologic examination showed mycosis fungoides.

DISCUSSION

DR. E. WILLIAM ABRAMOWITZ: There is always the possibility of coincidence. There is no reason why mycosis fungoides may not develop in a patient with psoriasis. I had a patient who had psoriasis for thirty years, proved several times to be psoriasis, in whom leukemia subsequently developed. There was no connection between the two conditions as far as I could see.

DR. TIMOTHY J. RIORDAN: It has been mentioned that this condition clinically resembled psoriasis, and I think it is worth while repeating an experience I had many years ago in Bellevue Hospital. The late Dr. Walter Highman examined a specimen from a case in which there was a clinical picture of psoriasis, and

the histologic picture was also of psoriasis. The patient returned a year later, and at that time the histologic picture was that of classic mycosis fungoides. In these cases of long drawn-out psoriasiform lesions, it is a good thing to keep in mind the diagnosis of mycosis fungoides.

DR. DAVID L. SATENSTEIN (by invitation): I think the fault in those cases is with the laboratory and not with the clinician. The changes seen in the epidermis are psoriasiform, and so the pathologists do not look any further. I had that happen to me until I had my attention called to the fact that in the reticular layer there are changes in the reticular cells which can be found with special stains. The changes in the cutis are important to search for. In the past few years I have been able to recognize mycosis fungoides long before the clinicians do. There is not much to say about the histologic observations in this case except that the slide showed what I like to present to students as the characteristic picture of mycosis fungoides. It showed everything that one would expect to find in this disease.

DR. TIMOTHY J. RIORDAN: Does Dr. Satenstein mean to say that all cases of psoriasis should have routinely an extensive study of the type he mentions?

DR. DAVID L. SATENSTEIN (by invitation): I may say that clinicians do not usually take histologic specimens in cases of ordinary psoriasis. If the condition is atypical, not behaving like psoriasis, a specimen is taken, and in such a case these careful studies should be made. I can show you many cases of mycosis fungoides in which the epidermis shows changes that look like psoriasis.

A Case for Diagnosis (Recurrent Ulcerations of the Mouth for Seventeen Years). Presented by DR. SAMUEL M. PECK.

H. B., a man aged 22, born in the United States, is presented. A painful ulcer first developed on the lower lip when he was 5 years old. Since that time, similar ulcerations of various sizes have appeared on the buccal mucosa, hard and soft palates, uvula, gums, tongue and pharynx. After regression the lesions leave contracted scars. In this process the uvula was destroyed four years ago. Several years ago superficial ulcerations in areas of excessive moisture, such as the axillas, groins and external genitalia, developed. About three years ago the lesions completely disappeared for one month after the patient had a temperature of 105 F.

General physical examination gives negative results except for the mucous membranes of the mouth. At the time of examination he presented ulcerations of various sizes beginning at the angle of the mouth and extending on to the buccal mucosa. Ulcers of similar character in other regions of the body, such as the groins, develop from raised erythematous patches with an irregular border; the edges, however, are always well defined. Such lesions measure from 1 to 1.5 cm. in diameter. Soon central ulceration develops, which rapidly involves the whole patch. The ulcer then increases in size, never extending deeply. The lesions heal with scarring. There is nothing to be seen at present aside from the oral lesions. In the mouth, all the stages can be seen. There are lesions on the inside of the lips and on the soft palate. The uvula and anterior pillars have been destroyed and replaced by scar tissue. The edges of the tongue are red and inflamed and appear irregularly punched out. The lesions bleed on the slightest trauma and are covered with a whitish exudate which is easily removed. Scars are seen in the groin. Only pea-sized lymph nodes can be felt along the cervical chain.

The Wassermann and Kahn reactions were negative. Blood count showed 91 per cent hemoglobin and 11,600 leukocytes per cubic millimeter, with 57 per cent segmented and 7 per cent nonsegmented polymorphonuclear leukocytes, 25 per cent lymphocytes, 4 per cent monocytes, 4 per cent eosinophils and 1 per cent basophils. Chemical examination of the blood showed 13 mg. urea nitrogen and 95 mg. sugar per hundred cubic centimeters. Cultures of the lesions in the groin showed Enterococcus and Staphylococcus albus. No higher bacteria or Ducrey bacilli were found. Cultures of lesions in the mouth showed Streptococcus

viridans A., Staph. albus D and Staphylococcus pharyngis siccus. Urinalysis gave normal results. A roentgenogram of the chest was normal.

Histologic examination of a lesion on the lip showed a chronic inflammatory reaction. The section showed parakeratosis and a rather atrophic epidermis. There was a dense inflammatory exudate extending from below the epidermis to the deep part of the cutis. It showed many dilated lymph spaces and thin-walled blood vessels. The inflammatory exudate consisted of a few polymorphonuclear leukocytes, many plasma cells, lymphocytes and eosinophils. Stains for bacteria, including the tubercle bacillus, were negative.

Histologic examination of tissue from the groin showed an ulceration extending from the epidermis to the upper part of the cutis. From the base of the ulcer there was an inflammatory process extending down to the subcutis. The epidermis at the margin of the ulceration was acanthotic and showed intercellular and intracellular edema. The floor of the ulcer showed nothing specific, just leukocytes, edema and dilated and congested vessels. Deeper toward the cutis many histiocytes were seen and rather compact masses of lymphocytes, among which were many plasma cells. The deeper blood vessels showed great thickening of their walls. This was especially true of the arterioles. The picture was that of a chronic ulcerative process and but for the absence of blood pigment was like the process seen in the vicinity of an ulcer on the leg. The diagnosis was ulceration suggestive of infection of unknown origin.

The patient has been treated with arsphenamine, a gold compound, germanin and antimony and potassium tartrate U. S. P. intravenously, with no effect. Sulfanilamide and sulfapyridine had a beneficial effect but had to be discontinued because leukopenia developed every time either drug was given. In the past the patient has had nicotinic acid, injections of liver, brewers' yeast by rectum, high vitamin diet, ultraviolet irradiation, all types of local antiseptics and hyperthermia, all without any benefit whatsoever.

DISCUSSION

DR. DAVID BLOOM: Has this condition been investigated from the standpoint of avitaminosis?

DR. SAMUEL M. PECK: Yes, intensive vitamin studies were done, especially when the patient was at the Rockefeller Institute. The patient is presented for diagnosis and for therapeutic suggestions. As you can see from the history, in spite of intensive study at the Rockefeller Institute, the Medical Center and Mount Sinai Hospital, the only diagnosis possible was a chronic inflammatory process.

DR. GEORGE C. ANDREWS: This man has never had a gastric analysis, and it would be a good idea to have one. He said he gets his chief relief from sodium bicarbonate. It makes his mouth feel better. Another case brought to mind by this one is that of a child of 5 years who had recurrent aphthous ulcers of the vulva for a period of about two years. The condition turned out to be Schüller-Christian disease.

DR. MAURICE J. COSTELLO: Since this case is presented for suggestions as to therapy, I should like to recommend the application three or four times a day of a cream made of zinc peroxide. It is possible that this condition might be caused by the microaerophilic streptococcus, as described by Dr. Frank Meleney, who also suggested this method of treatment.

DR. PAUL GROSS: Dr. Kesten and I had occasion to observe this patient at the Presbyterian Hospital a few years ago. We felt certain that we were dealing with a case of Sutton's periadenitis mucosa necrotica recurrens. Therapeutically, however, the patient remained a problem. After the introduction of a nasal tube, in order to avoid trauma to the buccal mucosa, an infection of the upper part of the respiratory tract developed, with high fever. Zinc peroxide was then used locally, and there was quick improvement. I feel that the credit for his improvement should be divided between zinc peroxide and the febrile reaction. I had hoped that fever therapy would give further beneficial results, but, as I have heard tonight, it proved to be unsuccessful. I have seen no beneficial results from

vitamin therapy. The identity of this condition with aphthous stomatitis cannot be entirely denied, and the possibility of virus disease, therefore, should be kept in mind. Zinc peroxide treatment was carried out by Dr. Meleney himself.

DR. FRANK C. COMBES: The lesions in the groin have apparently healed. What form of treatment was responsible for this?

DR. SAMUEL M. PECK: For the lesions in the groin I gave him antimony and potassium tartrate U. S. P. and a red mercuric sulfide (cinnabar) lotion. I cannot conceive of chronic inflammation in the mouth continuing into middle age without the possibility of an eventual development of a malignant condition. We treated this man with zinc peroxide, without results. The lesions he presented in the groins and axillas also began as papulopustules, just as the lesions in the mouth. It was significant that the lesions in the groins responded rather quickly to cinnabar lotion. They did well as long as they were kept dry.

Exudative Discoid and Lichenoid Chronic Dermatosis (Phase Resembling Leukotic Dyscrasia). Presented by DR. EUGENE T. BERNSTEIN.

S. I., a man aged 39, is presented from Mount Sinai Hospital, with an eruption of three and one-half years' duration, involving the entire integument, including that of the face and penis. These areas are covered with exudative, discoid and oval plaques or patches, from the size of a small pea to that of the palm. The shapes vary from discoid to larger elliptic and oval forms and present somewhat bizarre outlines. The lesions are elevated and sharply demarcated and show no tendency to central clearing. There is no vesiculation, no oozing and no crusting. The lichenification is the outstanding feature. The plaques have a tendency to follow the lines of cleavage. The glans penis shows a singularly persistent discoid plaque. In contrast to ordinary atopic dermatitis, the cubital and popliteal spaces are unaffected. The entire picture is dominated by the infiltration of the discoid and oval lesions, while lichenoid papules and crusting eczematoid elements are practically absent. While he was in Bellevue Hospital, a lymph node was excised from the right axilla, and a diagnosis of reticulum cell sarcoma was made.

Blood count showed 87 per cent hemoglobin and 9,800 leukocytes per cubic millimeter, with 58 per cent segmented forms, 15 per cent lymphocytes, 3 per cent nonsegmented forms, 3 per cent monocytes and 21 per cent eosinophils.

During the three and one-half years that this eruption has been present the patient has spent twenty-five months in various hospitals, without any relief. Salves which contain coal tar and are greasy give relief. The patient had an injection of diphtheria antitoxin eighteen months ago, and while he was in the febrile stage he was free of all lesions.

DISCUSSION

DR. FRANK C. COMBES: The patient has been under my care at Bellevue Hospital at intervals for over a year and has always presented a diagnostic and therapeutic problem. At times he was better, but his condition on discharge was essentially as it is at this time. The diagnosis of reticulum cell sarcoma was made by Dr. Symmers. It has never been clear to me what Dr. Symmers' interpretation of this condition is. We have had several patients for whom a similar diagnosis has been made on the basis of the observations on the excised lymph nodes. Some of these patients have recovered, either spontaneously or after roentgenotherapy of the lymph nodes. There is a question of contact dermatitis in this patient also. The condition began on his hands and spread to other parts of his body. If I recall correctly, he worked with metal filings, to which he attributed the initial dermatitis.

DR. TIMOTHY J. RIORDAN: The description of this condition given by Dr. Symmers is that of multiple giant hyperplasia of the lymph follicles. He says some of the cells suggest reticulum cell lymphosarcoma.

DR. FRANK C. COMBES: At the time of hospitalization at Bellevue, this patient did have considerable lymphadenopathy. He did a great deal of scratching and was covered with excoriations. This lymphatic enlargement could be ascribed to the chronic pruritus. The inguinal nodes were the size of a fist. Now they are much smaller. He also had an enlarged gland in his right breast which disappeared after roentgenotherapy.

DR. DAVID BLOOM: I am glad to hear that the diagnosis of reticulum cell sarcoma is not being taken seriously by the staff of Bellevue Hospital. However, I have observed cases in other hospitals where the diagnosis of reticulum cell sarcoma carried from Bellevue Hospital was taken seriously. I believe that it is important to have another biopsy done of the lymph nodes and examined in a different laboratory, in order to determine the diagnosis.

DR. MAURICE J. COSTELLO: I should also like to clarify the diagnosis of giant follicular lymphadenopathy with or without splenomegaly, as described by D. Symmers and W. Hutcheson (*Arch. Path.* **26**:603-647 [Sept.] 1938). A number of patients suffering from generalized pruritic eczematous eruptions have had this diagnosis made, based on a histologic examination of the lymph nodes. Observations over a period of three or four years have shown that many recovered completely from the eruption and the accompanying lymphatic glandular enlargement. I am under the impression that the lymphatic glandular enlargement is secondary to the irritation caused by scratching.

DR. PAUL GROSS: Many patients with this condition have a history suggesting contact dermatitis at the onset, and occasionally one can observe the transition into the chronic exudative picture.

DR. EUGENE T. BERNSTEIN: The concept of this peculiar and distinct dermatosis was inaugurated by Dr. Isadore Rosen, and the syndrome was first described by Sulzberger and Garbe (ARCH. DERMAT. & SYPH. **36**:247 [Aug.] 1937) in a report of a group of 9 cases.

The characteristics of this eruption can be reduced to four phases: (1) the exudative and discoid phase, (2) the lichenoid phase, (3) the urticarial phase and (4) the phase resembling the prefungoid stage of mycosis fungoides. The last is the one presented here tonight, resembling a leukocytic dyscrasia. The case is not an instance of mycosis fungoides, because there is involvement of the entire integument, including the face and scrotum; the majority of lesions consist of exudative discoid plaques, and they present irregular and somewhat bizarre outlines. They are fairly sharply demarcated and do not show any tendency to central clearing. There is practically no erythema. The crusts covering the lesions are of the serous type. The glans penis presents a persistent discoid edematous plaque. The eyelids and the external ears are practically free. On the trunk the long axis of the oval plaques shows a distinct tendency to follow the lines of cleavage, so characteristic of pityriasis rosea. The most characteristic lesions are the sharply demarcated oval and discoid plaques showing variations in consistency and appearance; these plaques are at times flat and scaly, at times elevated and edematous, and at times crusted; they speedily pass through many of the clinical stages considered characteristic of eczematous lesions.

In regard to the report from Bellevue Hospital of reticulum cell sarcoma or Symmers' disease, there has not been any progression in the lymph nodes. I plan to perform another biopsy of the lymph nodes and of the skin. It has been observed that this disorder occurs mostly in middle-aged Jewish persons, and it seems that there must be some endocrine gland disturbance.

DR. OSCAR L. LEVIN: This type of eruption is common. It has been described under various names. The title as presented this evening merely describes the clinical picture now present. The cause of the eruption is obscure. It may indicate a contact dermatitis, an occupational dermatitis or an obscure or unidentified generalized dermatosis; it may be a beginning lymphoblastoma. I know of 1 case which had been reported as belonging in this group and which merely presented secondary changes of previously existing dermatitis herpetiformis. In regard to Symmers' disease, which has been mentioned this evening, I may describe briefly

a patient who originally presented a picture similar to that seen in the patient presented this evening and in whom generalized adenopathy developed and subsequently an eruption, which took on the characteristics of mycosis fungoides. The patient finally died with an eruption of universal erythroderma with tumor formations and ulcerations, and biopsy revealed evidence of a disease of the reticulo-endothelial system and pathologic changes of the skin suggestive of lymphosarcoma. These tumors were also found in the internal organs.

Poikilodermatomyositis (Petges-Clijat). Presented by DR. PAUL GROSS.

A. B., a woman aged 30, born in Puerto Rico, a resident of the United States for the past thirteen years, began to complain about a year and a half ago of pains in the left arm and leg. The pain was accompanied by redness and tenderness of the muscles. Later she complained of pain in the back, headaches and frequent nosebleeds and felt very weak. She then noticed a swelling and discoloration of the eyelids. By the end of April 1939 the pains had become severe. She lost her appetite and frequently felt nauseated. Within one year she had lost 19 pounds (8.6 Kg.) in weight.

On May 8, 1939, the patient was admitted to the Hospital for Joint Diseases. At that time examination showed that the lids of both eyes, especially the lower lids, were puffy, bluish red and discolored. The redness extended to the zygomatic and temporal regions, where more pronounced pigmentation showed. On both elbows there were circumscribed irregular patches of red atrophic skin of harsh consistency. Within these areas there were telangiectases and deep pigmentation, as well as slight scale formation. There were similar patches with less pronounced atrophy over the knees. On the small joints of both hands there were circumscribed patches of atrophy, some resembling lupus erythematosus, and a bandlike region extending from the right elbow to the wrist, showing moderately advanced changes. There was another area of slight atrophy and pigmentation near the coccygeal region. The muscles of the arms, forearms and hands showed considerable wasting and had a stringlike consistency. The ulnar nerves were enlarged. Neurologic examination showed the left knee jerk to be more active than the right and the achilles tendon reflexes to be diminished. There was some hypalgesia over the left lower extremity, left hand and forearm. On the posterior surface of the right thigh there was redness and deep-seated tenderness (acute myositis).

The Kahn reaction was negative. Urinalysis gave normal reactions. A blood count showed 11.5 Gm. of hemoglobin per hundred cubic centimeters and 3,840,000 erythrocytes and 5,200 leukocytes per cubic millimeter, with a differential count of 71 per cent segmented neutrophils, 2 per cent eosinophils and 2 per cent lymphoblasts. A nasal smear for lepra bacilli was negative. The sedimentation rate of the blood was 40 mm. in forty-five minutes. Agglutination for the hemolytic streptococcus was positive with dilutions up to 1:280. The determination of cozymase by the factor V of Haemophilus influenzae gave normal values. Chemical examination of the blood showed 4.3 mg. albumin, 10.5 mg. calcium, 4.2 mg. globulin and 3.8 mg. inorganic phosphorus per hundred cubic centimeters and an albumin-globulin ratio of 1+. Gastric analysis showed the following results on a fasting stomach and in one, two, three and four hours after administration of histamine, respectively: total acids, 14, 21, 16, quantity not sufficient, 18; total free acids, 0, 17, 10, quantity not sufficient, quantity not sufficient; free hydrochloric acid, 0, 14, 6, quantity not sufficient, 7. The result of a dextrose tolerance test was normal. A roentgenogram of the skull showed clouding of the left antrum and frontal sinus. Roentgenograms of the bony skeleton showed calcification of the soft tissue in both axillas. The sella turcica was normal.

Histologic examination (by courtesy of Dr. J. Gardner Hopkins) showed the following: A section of the skin showed a chronic inflammation in the subepidermal layer and perivascular lymphocytic infiltration. There was fragmentation of the collagen fibers in the subpapillary layer, and a few dilated capillaries were seen

in the papillae. The epidermis was normal except for slight *alteration cavitaire*. A section of muscle from the area of more acute myositis of the thigh showed islets of lymphocytic infiltration between the muscle bundles. There was hyaline degeneration of the muscle bundles in special areas and fibrillary degeneration. In one of the hyalinized muscles there was a blood vessel with a thickened wall.

The course in the hospital was afebrile. Under intensive therapy with liver extract, nicotinic acid and thiamine, the patient's general condition improved. The acute myositis of the thigh disappeared. There was a moderate gain in weight, and the patient was able to walk again, but there was no change in the muscular atrophy. The patient has been ambulatory since that time and has not had any attack of acute myositis.

I have attempted to investigate this case thoroughly. The only positive fact is an improvement of the patient, who was unable to walk on admission to the hospital, under treatment with liver extract, nicotinic acid and thiamine.

DISCUSSION

DR. CHARLES WOLF: This condition is rare, of course, but the diagnosis is made by the poikiloderma-like changes in the skin associated with myositis, which in this particular case I fail to see. However, she has a symptom complex which might be well entertained, associated with another condition, of which I have seen two examples. She has a sclerodermatous change in the skin and subcutaneous tissues and also a moderately high calcium content of the blood. I should therefore like to make a tentative diagnosis of hyperparathyroidism. Cases like this have been observed at Mount Sinai Hospital, in which operation has showed adenomas of the parathyroid gland to exist.

DR. OSCAR L. LEVIN: I agree with Dr. Wolf in his discussion of this patient's condition. The lesions present are those of scleroderma associated with deposits of calcium, and there are areas presenting lesions of poikiloderma as well as lesions suggestive of myositis. The condition may have begun as the result of an acute infection, and the picture as now presented probably depends on dysfunction of the endocrine system, particularly in the parathyroid glands, as indicated by calcium deposits.

DR. EUGENE T. BERNSTEIN: There are four types of poikiloderma, namely, (1) poikiloderma atrophicans vasculare (Petges and Jacobi type), (2) reticulated pigmented poikiloderma of the face and neck (Civatte type), (3) poikiloderma associated with muscular sclerosis (G. Petges type) and (4) poikiloderma associated with sclerodactylia (Petges and Clijat type). All these types are, in the opinions of the authors, preponderantly of endocrine origin. Civatte, Milian and Perin, Gilchrist, Bowman and Clark expressed the opinion that the condition is a consequence of adrenal insufficiency (Bowman, K. A., and Clark, E. C.: ARCH. DERMAT. & SYPH. **15**:583 [May] 1927). Brescovsky, Photinos, Bloch and Little said that dysfunction of the ovaries and of the parasympathetic nervous system is responsible for poikiloderma (Bloch, B., and Stauffer, H.: ARCH. DERMAT. & SYPH. **19**:22 [Jan.] 1929). It is interesting here to study the results of chemical examination of the blood, especially the calcium content. As a therapeutic measure, I suggest the administration of mixed glandular extracts.

DR. FRANK C. COMBES: I agree with the diagnosis as presented. Disturbances in calcium metabolism are not infrequently noted in cases of dermatomyositis. This patient does have poikiloderma. There is a good-sized patch on the back of the neck, extending over the shoulders. The entire condition may be due to parathyroid disease, as already mentioned.

DR. PAUL GROSS: I am unable to see any typical lesions of scleroderma in this patient. On the other hand, most of the lesions show the features of poikiloderma, which, of course, implies a peculiar harshness of the skin, telangiectases, pigmentation and atrophy. Although this patient has definite deposits of calcium in the muscles, the calcium content of the blood is not high. The most significant feature of these cases is their questionable relation to poikiloderma vasculare

atrophicans of Jacobi. G. B. Dowling and W. Freudenthal (*Brit. J. Dermat.* **50**:519 [Oct.] 1938) try to differentiate between poikiloderma of Jacobi and dermatomyositis on clinical and histologic grounds. The histologic examination of the skin showed a decided inflammatory reaction, which is probably due to the fact that the specimen was taken from an early lesion.

Darier's Disease. Presented by DR. ISADORE ROSEN.

J. D., a man aged 43, is presented from the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital, complaining of an eruption of fifteen years' duration involving the trunk, arms and legs. Examination shows pinhead-sized and larger, brownish or reddish papules, covered by a thin scale. These are grouped, forming large areas on the trunk, more pronounced on the lower part of the chest and sides of the chest. There are scattered lesions on the trunk, arms and legs. Behind the ears are some verrucous lesions, and on the lower part of the legs are eczematous patches.

The patient received antisyphilitic treatment from 1917 to 1932. Fifteen years ago he received roentgenotherapy.

The Wassermann and Kahn reactions were negative. The histologic diagnosis was Darier's disease. The epidermis showed acanthosis, mainly of the rete pegs, with digitate proliferation of the rete pegs in areas. In the epidermis there were dyskeratosis and corps ronds. The horny layer in the dyskeratotic areas was considerably increased. In the subepidermic zone in these areas there was a moderate amount of simple infiltrate.

DISCUSSION

DR. EUGENE T. BERNSTEIN: I should like to call attention to the facial expression of these patients with Darier's disease. The majority of them have a sullen and disagreeable expression. The epidermis and the central nervous system develop from the same germinal layer. Injuries to the ectoderm during fetal life may produce simultaneous occurrence of cutaneous malformations and mental abnormalities. This is true in cases of Recklinghausen's disease, adenoma sebaceum, cutis verticis gyrata and, as in this case, Darier's disease.

A Case for Diagnosis (Hereditary Familial Telangiectasia?). Presented by DR. MAX SCHEER.

T. U., a girl aged 9 years, is presented from Mount Sinai Hospital. About three years ago she had a tonsillectomy, and the mother states that the lesions began to appear at about that time. The eruption consists of areas of telangiectasia, varying in size from that of a pinhead to 5 mm. in diameter, located on the dorsum of the nose, the left cheek and left side of the upper lip, with one or two on the upper part of the back. The patient has had severe nosebleeds at irregular intervals, the last one about one week ago.

Physical examination shows no abnormalities except the lesions aforementioned. There are no lesions in the nose or pharynx or on the tongue. No history of a similar condition in the family can be obtained. The Wassermann reaction was negative.

DISCUSSION

DR. DAVID BLOOM: The lesions in this case are located on one side of the face, and it is more probable that the condition is a nevus rather than hereditary telangiectases.

Dermatolysis (Ehlers-Danlos Syndrome). Presented by DR. ABRAHAM WALZER.

A. R., a boy aged 3, is presented for Dr. Jacob Skeer, from the Department of Dermatology of Brooklyn Jewish Hospital. He was first seen in the Pediatric Clinic of the hospital by Dr. Arthur Kaplan and complained of excessive bruising

and bleeding. The family history was irrelevant. The child was a premature infant (thirty-two weeks' gestation) and weighed only 2½ pounds (1.13 Kg.) at birth. His development was normal and also the feeding history. The present condition was first noticed when he began to walk at approximately 14 months of age. The child bruised easily, and ecchymoses developed as a result of minimal trauma. When he suffered an open wound the bleeding was excessive and required tight dressings to produce hemostasis. He did not experience any spontaneous hemorrhages. No bleeding was ever noted from the mucous membranes.

Physical examination shows a friendly, well nourished and well developed child. The positive findings are limited to the skin and joints. The skin is velvety, with great hyperelasticity. It is dry and scaly and of doughy consistency. There are linear, irregularly shaped and sized scars on the forehead and two scars on the right cheek. On the right side of the neck there is a scar resulting from an operation for abscess. There is an elliptic scar, ivy leaf in size, 1½ inches (3.8 cm.) below the left scapula. Over the left tibia is a scar caused by the removal of a section for histologic study. There are irregularly shaped, multilobular, violaceous, soft, raisin-like tumors and subcutaneous nodules, as well as flat depressed atrophic scars on both elbows. These are hyperpigmented on the margins. Manipulation of the joints shows a moderate degree of hyperflexibility. The muscles are somewhat flabby.

Blood count showed 81 per cent hemoglobin, 4,300,000 erythrocytes and 13,000 leukocytes per cubic millimeter, with a differential count of 40 per cent polymorphonuclear leukocytes, 40 per cent lymphocytes, 16 per cent eosinophils and 4 per cent monocytes. There were no pathologic cells. Bleeding time was one minute and clotting time seven minutes. The results of bone marrow puncture confirmed the peripheral eosinophilia. Urinalysis gave normal results. The Kline and Mantoux tests gave negative results. The clot retraction time was eight hours, with a normal clot. Roentgenograms of the long bones and of the lungs were normal. Chemical examination of the blood showed 587 mg. total lipids, 203 mg. total cholesterol (28 per cent free cholesterol), 11.2 mg. calcium, 4.6 mg. phosphorus and 16.9 units phosphatase (Bodansky) per hundred cubic centimeters. Chemical examination of the skin showed total lipids 32.1 per cent (of dry weight), total cholesterol 0.48 per cent and total solids 52.4 per cent.

Histologic examination by Dr. David L. Satenstein showed the following changes: The horny layer was thickened and filled in the dilated follicular openings. The epidermis presented no abnormalities. Most of the cutis framework was arranged parallel to the surface, in bundles, not sclerosed. The vessels in the subepidermic zone were in groups of three and four, all of the same size (telangiectatic). The connective tissue network of the fat zone was somewhat thicker than normal. Some of the walls of the smaller vessels were swollen. Perivascular lymphatic spaces were dilated and contained few cells. The intima of the large deep vessels had exfoliated into the lumen. The elastica was everywhere present and apparently increased in amount. The subepidermic network was dense. The fibers of the middle and deep portions of the cutis were much thicker than normal.

Leukoderma Acquisitum Centrifugum (Sutton). Presented by DR. HENRY D. NILES.

F. S., a girl aged 13, is presented from the Skin and Cancer Unit of the New York Post-Graduate Medical School and Hospital, where she was first admitted in July 1939. At that time she had several small to large pinhead-sized, soft, raised, round, darkly pigmented nevi, with a surrounding ring of leukoderma scattered over the face, neck, back and upper part of the chest. Since her first visit to the hospital, most of the nevi have been destroyed by electrodesiccation. In most of the lesions the surrounding area of leukoderma has become much fainter. There are still two or three typical untreated lesions on her back.

The patient is presented to demonstrate the return of pigmentation in the leukodermic area after the destruction of the central pigmented nevus.

Urticaria from Heat. Presented by DR. MAX SCHEER.

R. G., a woman aged 22, is presented from Mount Sinai Hospital. She complains of itching and swelling of the lips and tongue and substernal sensation of pressure following ingestion of hot foods. She states that exposure to sunlight produces wheals. The duration is eight months.

Physical examination gave negative results. Wheals can be produced by the application of a test tube filled with hot water or by exposure to a 100 watt electric light bulb at a distance of 6 inches (15 cm.) for two minutes.

Treatment with histaminase by mouth for two weeks was ineffectual. At present the patient is receiving histamine (0.8 cc. of 1 to 1,000 solution). She has had about ten injections, with little benefit so far.

The Wassermann reaction was negative.

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Jan. 19, 1940

Hypertrophic Nevus of the Scalp, Face and Oral Mucosa. Presented by DR. CARROLL S. WRIGHT.

J. S., a white man aged 20, presents extensive coalescent patches of cauliflower-like hypertrophic and verrucous epithelial growth involving most of the scalp, particularly the left half. This tendency to hyperkeratotic overgrowth is also present on the left side of the forehead and on both sides of the face, occurring here and there in linear bands. There is also involvement of the left ear, left upper eyelid, left half of both lips and left half of the hard and soft palate and both halves of the tongue. Within the past five weeks a small vegetative patch of hyperkeratosis has appeared in the interscapular region. Serologic tests of the blood have given negative results. About fifteen years ago the chin, the under surface of the jaw and the right half of the neck were treated "with the electric needle." This resulted in scar formation in those regions six years ago.

DISCUSSION

DR. DONALD M. PILLSBURY: Although roentgen ray therapy is not of value ordinarily for nevi unless there is much angiomatous tissue, it seems to me that this nevus, which has such a greasy character, indicating considerable sebaceous hypertrophy, may be benefited by roentgen rays. This treatment undoubtedly will not clear the lesion greatly, but it might lessen the greasy character and the verrucous hypertrophic tissue.

DR. ABRAM STRAUSS: I agree with Dr. Pillsbury, but I suggest the use of the Chaoul tube (5,000 to 9,000 r confined to a small area). I have seen remarkable results in nevi of different types from this therapy.

DR. JOSEPH V. KLAUDER: I obtained excellent cosmetic results in a similar case with electrodesiccation (Klauder, J. V.: The Treatment of Nevi, *J. A. M. A.* **90**:1763 [June 2] 1928). The operation in this case was performed in three stages, one third of the lesion being removed at each stage, with the patient under nitrogen monoxide anesthesia, followed by irradiation of the keloidal scar.

DR. CARROLL S. WRIGHT: According to the history, some one has tried desiccation on a small area of this lesion. That was the method of treatment I suggested when I first saw the patient.

Recurrent Erythema Multiforme of the Oral Mucosa Associated with Progressive Fordyce's Disease. Presented by DR. REUBEN FRIEDMAN.

L. M., a white man aged 22, has since March 1936 been subject to recurrent attacks of painful ulcerations of the oral mucosa, which necessitated hospitalization on five occasions. Each attack lasted about two months. In the present attack, which began in February 1939, he has been going through cycles of exacerbation and remission, without, however, at any time becoming entirely free from erosive lesions. When he was presented before the meeting of the American Academy of Dermatology and Syphilology at Philadelphia on November 7 he was in a stage of remission, exhibiting only one erosive lesion on the lower lip. About December 15 acute exacerbation began, and now he presents numerous ulcerative lesions of the labial, buccal and lingual mucosa. For the past week he has had fever, with a temperature as high as 101.4 F. At no time during the past year has he shown any cutaneous involvement. The lesions of Fordyce's disease on his lips and buccal mucosa are more prominent and greater in number today than they were a year ago.

DISCUSSION

DR. FRED D. WEIDMAN: By "progressive" does Dr. Friedman mean that there was an increase in the number of lesions or that the preexisting lesions became larger?

DR. REUBEN FRIEDMAN: I mean a definite increase in the number. This patient has been under my observation for over two years, and I am convinced that during those two years there have appeared more patches of Fordyce lesions on his labial and buccal mucosa than there were in the first months that the patient was under my care. I realize that this is not in agreement with our conceptions of the nature of Fordyce lesions anatomically, but I explain it on the basis that the recurrent inflammatory involvement of the labial and oral mucous membranes has brought to the surface aberrant sebaceous glands that were not previously visible.

DR. ABRAM STRAUSS: This case may be one of beginning of pemphigus.

DR. JOSEPH V. KLAUDER: Has he ever had lesions elsewhere?

DR. REUBEN FRIEDMAN: He had, about a year and a half or two years ago, five or six small vesicles on the backs of the forearms and hands. I thought they were evidence of beginning pemphigus. My first diagnosis in this case was pemphigus confined to the oral mucosa, and I anticipated cutaneous involvement. But he has never since shown other cutaneous, conjunctival or nasal mucosal involvement.

DR. SIGMUND S. GREENBAUM: This man was a patient of mine at the Philadelphia General Hospital some time ago. Because of the history and clinical appearance of the eruption, I made a diagnosis of recurrent erythema multiforme but could not determine the causative factor. I do not recall any observation of erythema multiforme terminating in pemphigus vulgaris. I have, however, observed typical dermatitis herpetiformis terminate in pemphigus vulgaris. In connection with the oral condition of this patient, I want to call your attention to two items: a scar, 1 cm. in diameter, on the left side of the soft palate near the anterior pillar and leukokeratosis of the buccal mucosa, which is frequently observed with acute inflammatory conditions of the oral mucosa. One may see the latter condition around secondary syphilids; it disappears when the acute lesions fade.

A Case for Diagnosis (Erythroplasia of Queyrat?). Presented by DR. REUBEN FRIEDMAN.

N. R., a white man aged 38, presents a number of sharply defined, superficial, painless, glazed or moist, erosive and slightly crusted patches of dermatitis on and about the glans, corona, meatus and shaft of the penis. When the lesions are wiped dry, tiny droplets of clear fluid appear immediately on some of them. On palpation the lesions show no infiltration, although the consistency of some

of them is slightly greater than that of the surrounding skin. There is and has been no associated inguinal adenitis. The condition began in July 1938 as a single erosive lesion in the right coronal sulcus, and new lesions thereafter appeared from time to time. Some have undergone spontaneous involution. In the main, however, the condition has been unresponsive to local therapy, including the application of various ointments and wet dressings, roentgen ray therapy and cauterization with dichloroacetic acid. Small doses of neoarsphenamine and intravenous injections of a bismuth compound have been tried, with no benefit. There is no history of the ingestion of drugs or of venereal disease. Dark field examination and serologic tests of the blood have given negative results. The patient has been subject in the past year to recurrent outbreaks of a mild eczematous dermatitis on the arms and legs, of uncertain cause but apparently readily responsive to local therapy.

DISCUSSION

DR. HERMAN BEERMAN: I think that it will eventually be found that the patient has one of those conditions which Dr. Sulzberger and Dr. Garbe described as distinctive exudative discoid and lichenoid dermatosis (ARCH. DERMAT. & SYPH. **36**:247 [Aug.] 1937). I had a similar case some time ago in which Dr. Gross of New York made the prediction that generalized lesions would appear. The prediction came true. When Dr. Sulzberger was here he paid special attention to the localization of this condition to the penis and the tip of the nose in the early phases.

DR. ERICH URBACH (by invitation): I am wondering whether this condition is the beginning of Bowen's disease.

DR. DONALD M. PILLSBURY: Has a biopsy been performed?

DR. REUBEN FRIEDMAN: Not yet, but the patient granted permission today. So far as the suggestion of an exudative discoid and lichenoid dermatosis of Sulzberger and Garbe is concerned, this patient has had recurrent eczematous lesions on his extremities and also on the tip of the nose, as Dr. Beerman mentioned. On the other hand, he has had this condition on the penis for a year and a half; so it could hardly be incipient dermatosis of Sulzberger and Garbe with an incubation of a year and a half. Perhaps a biopsy will throw more light on the question. I presented this case as one for diagnosis because of the coexistence of the eczematous lesions recurring over a period of a year and a half and the lesions on the penis, which simulated erythroplasia of Queyrat (Sulzberger, M. B., and Satenstein, D. L.: ARCH. DERMAT. & SYPH. **28**:798 [Dec.] 1933).

DR. DONALD M. PILLSBURY: I believe the lesions on the body are not particularly eczematoid tonight. They are dry and psoriasiform. I have observed 1 case of psoriasis on the glans penis in which the condition looked much like this.

DR. SIGMUND S. GREENBAUM: I should like to suggest that a biopsy specimen be taken not only from the most active part of the penile lesion but also from the active lesion on the left side of the chest.

DR. REUBEN FRIEDMAN: The lesions at no time were psoriatic. They always presented crusted and vesicular elements.

Imbibitio Lipoidica Collageni Degenerati Cutis. Presented by DR. ERICH URBACH (by invitation).

E. G., a white woman aged 38, apparently in good health, presents two types of lesions. The majority, about fourteen, are depressed in the center, 5 mm. in size, brown red, of cartilaginous consistency and deep seated and are located on the back, the lower part of the abdomen, the left buttock and the legs. The remainder (type 2) are elevated, 10 mm. in size and hard and are situated above the right patella, below the left patella (removed for biopsy) and on the outer surface of the left ankle. The first lesion appeared twenty-six years ago on the extensor surface of the right thigh. The others developed in the last two to eight years. The total cholesterol content of the blood was 160 mg. per hundred

cubic centimeters, and the cholesterol esters were 50 mg. per hundred cubic centimeters. The dextrose tolerance test gave a normal result. A histologic study was made of the specimen of eight years' duration. With hematoxylin and eosin stain, atrophy of the stratum corneum and proliferation of the connective tissue cells were seen. The latter showed vesicular shaped nuclei. Between the fibrous tissue cells there were numerous longitudinal spaces, apparently due to the extraction of a fatlike substance during the treatment of the sections with alcohol. There was thickening of the walls of the vessels. With sudan IV stain the stratum reticulare at low magnification became brown red. At higher magnification, globules and granules appeared to be arranged in series, especially between and in the fibrils of the connective tissue. Typical foam cells could not be demonstrated. Some histiocytes were filled with fine fat granules. Double-refractive bodies were not demonstrable. The lesions of two years' duration showed considerable proliferation of the connective tissue without lipoid imbibition.

DISCUSSION

DR. FRED D. WEIDMAN: Dr. Urbach has given me two previews of this case, and I have studied the sections in the laboratory. He is presenting this case as one of a clinical entity, that is, of imbibitio lipoidica collageni degenerati cutis. The histologic sections, in my opinion, show the architecture of a fibroma, and my feeling when I saw the sections was that the architecture was essentially fibromatous and that the xantomatous changes were secondary. That is in line with other lesions that are known on the skin; scars, for example, behave in this way. After seeing the patient tonight clinically, I still think that this is most likely the case. The patient has indiscriminately distributed lesions of various ages. Some are so old that there is no infiltration at all: just a brown macule. Fresher lesions that are infiltrated show the character of dermatofibroma lenticulare. In my opinion "imbibitio" is a proper designation for the yellowing phase of the pathologic process present, but the fundamental and primary change is the fibromatous one. The general pathologist always designates first of all the fundamental pathologic process, and if there are secondary changes, he adds them as an appendix. Thus, the way in which I view this condition is as a dermatofibroma lenticulare with secondary xanthomatous or lipoidal changes.

DR. SIGMUND S. GREENBAUM: I did not see the histologic sections, but on clinical grounds alone I agree with Dr. Weidman that the condition is fibroma durum or subepidermal nodular fibrosis. I have presented several cases of this condition at previous meetings of this society.

DR. ERICH URBACH (by invitation): There is no question that this disease is primarily a fibroma or dermatofibroma lenticulare and that the changes observed appear secondarily. The term "imbibitio" means that the condition is not a primary disease, e. g., a so-called infiltration. I am opposed to calling it a fibroxanthoma because in this type of case, of which I have observed 3, one cannot find so-called xanthoma cells (foam cells). In these cases, primarily in the lesions which are two to five years old, only the histologic picture of fibroma is shown; and then after the lesions are older, eight, ten or twenty years old, a lipoid material appears between the fibrils or in the fibrils. This lipoid is different from that of xanthelasma, because it does not produce double refraction and because it is soluble in ether and in boiling hot acetone, largely but not completely soluble in cold acetone and in cold and boiling hot absolute alcohol and insoluble in chloroform. If names were constructed only from the clinical appearance, one would also not be justified in speaking of necrobiosis lipoidica, because this name refers to pathologic rather than to clinical changes. In spite of this the name has been accepted. I believe Dr. Weidman is absolutely right if he believes that the lipoid changes are secondary. In 2 other cases I was able to make extensive studies of the lipoid constituents of the blood, which revealed a decided disturbance of the fat metabolism.

DR. SIGMUND S. GREENBAUM: In the 8 or 9 cases of fibroma durum which have come under my observation in recent years, the clinical picture has been

that of a spontaneous development of one or more hard nodules, 1 mm. to 8 mm. or larger, located particularly on the lower extremities. They persist for a variable period, sometimes many years, and have in many instances undergone spontaneous regression, usually leaving a pigmentary, slightly atrophic area. I might add that Darier (Darier, J.: Précis de dermatologie, ed. 4, Paris, Masson & Cie, 1928, p. 1000) noted xanthomatous, mucoid and even calcareous degeneration occurring in some of these fibromas.

Geometric Phagedena (Brocq). Presented by DR. SIGMUND S. GREENBAUM.

In H. T., a white man aged 21, an ulcer developed on the left leg in 1933, after an injury. One week later a similar lesion appeared on the opposite leg. These healed only one year after skin grafts. The site on the right leg broke down again about fourteen months ago. It eventually healed after skin grafts were applied, but the region on the anterior part of the right thigh from which the graft was taken ulcerated and has been resistant to treatment to the present time.

The patient appears somewhat undernourished but not actually ill. A palm-sized ulcer with gyrate, undermined, rolled borders is present on the anterior aspect of the right thigh. Parchment-like scars are found on the lower half of both legs at the sites of the healed ulcers and also on the right deltoid region, where previous vaccination and autoinoculation resulted in "takes." There is also a superficial eczematoid dermatitis between the toes and on the dorsa of both feet. There is a soft, blowing diastolic murmur heard over the precordium. The remainder of the medical examination gave negative results. Repeated cultures from the lesions and from active autoinoculative sites revealed nonhemolytic streptococcus and hemolytic Staphylococcus albus. Autoinoculation and injection of combined viable cultures resulted in "takes." Injection of each organism separately yielded negative results. Repeated Wassermann reactions of the blood were negative. The ulcer was treated by excision, injections of vaccine, and nonspecific protein therapy, by the local application of bacterial antigen and of antiseptics, with allantoin, urea, zinc peroxide, gentian violet, sulfanilamide, sulfapyridine, roentgen rays, and dichloramine-T, by irrigations with potassium permanganate, and by ultraviolet and infra-red irradiation.

DISCUSSION

DR. DONALD M. PILLSBURY: Is this not the first instance in which the symbiotic nature of the infection has been proved in man?

DR. SIGMUND S. GREENBAUM: For this rare type of phagedena, Brocq in 1916 proposed the term geometricum. It is certainly descriptive of the clinical picture of this type of chronic pyoderma, as can be easily observed in this case. In 1930 Brunsting, Goeckerman and O'Leary (ARCH. DERMAT. & SYPH. **22**:655 [Oct.] 1930) described 5 cases of what appears to be the same condition but which they termed pyoderma (echthyma) gangraenosum. More recently Montgomery (ARCH. DERMAT. & SYPH. **40**:924 [Dec.] 1939) described a similar case under the same designation. In Brunsting's cases nonhemolytic streptococci and staphylococci were cultured more or less uniformly, and he was able to produce a reaction in rabbits when cultures of both organisms were injected at the same point and together in the rabbit's skin. The organisms injected separately failed to produce disease. I have observed since 1922 about 7 patients with geometric phagedena. In my first case, the ulcerative lesion had involved the entire abdominal wall from the pubic bone and the iliums below the ribs and the xiphoid process of the sternum above. In 2 of these cases I was able to produce in the patient's skin elsewhere lesions similar to the original lesion by injecting at the same point living cultures of the organisms obtained from the primary ulcerative lesion. In these 2 cases two different types of organisms had been obtained from the active lesions. The streptococcus was, however, constant. When the living liquid cultures (1 minim, 0.06 cc.) were injected separately, no reaction at the site of the injection appeared. When both organisms were injected (1 minim [0.06 cc.] of each living culture was mixed and 1 minim [0.06 cc.] of the resulting mixture

used for the test), reaction took place in the form of the primary ulcer. This work was done in 1933, and the material, including photographs, was presented in the round table discussion at the first meeting of the American Academy of Dermatology and Syphilology in St. Louis (ARCH. DERMAT. & SYPH. **40**:121 [July] 1939). So far as I know, this is the first experimental proof in man of a symbiosis which has been shown to be the chief factor in the production of certain of these ulcers. In connection with the present case, I want to call attention to the site of the autoinoculation test, which is now healed. Pus from the original lesion was rubbed into a superficial abrasion of the skin over the right deltoid region. On the third day slight inflammation took place, and by the fourth day a pustule covering a definite ulcer, similar to what one sees in the autoinoculation chancroid, developed. The autoinoculation chancroid, however, develops within twenty-four to forty-eight hours. The smears and cultures from the autoinoculation pustule were studied for Ducrey bacilli, but only streptococci were found. An Ito-Reenstierna chancroidal test performed subsequently gave a negative result. An interesting feature in connection with this man's ulcer is that a week after he was admitted to the hospital typical erysipelas developed, beginning at the lower border of the ulcer, extending within three days well below the knee and accompanied by toxemia and high temperature. This erysipelas, as well as the ulcer, failed to improve under sulfanilamide therapy, but the high temperature induced by the erysipelas fell to normal within twenty-four hours after the ingestion of 60 grains (3.88 Gm.) of sulfapyridine. The interesting point, of course, is that the streptococcus in the ulcer was able to produce a severe erysipelas but without ulceration at the site of the erysipelas, such as was present in the original lesion, all of which points still more to a symbiosis as the factor in the phagedenic ulcer. In 1 of my cases repeated cultures made at two different institutions were persistently negative. In another patient the streptococcus and Bacillus proteus were recovered. When these, in the form of living cultures (1 minim, [0.06 cc.]) were tested separately by intracutaneous injection into the patient, there was no reaction when they were mixed (1 minim [0.06 cc.] of a 1 to 1 mixture of the two cultures) and then injected, a lesion similar to the original ulcer developed. In a third case, already noted, streptococcus and staphylococcus were recovered, and when they were injected together in living cultures an ulcer similar to the primary ulcer developed. At the present time, therefore, I find it difficult to believe that the same organisms are always the factors in the production of phagedenic ulcers, but at least two things appear to be certain: One is that a streptococcus is constant, as Brunsting and others before him have shown, and the second is that these lesions are produced by symbiosis.

DR. JOSEPH V. KLAUDER: How does this differ from tropical ulcers?

DR. SIGMUND S. GREENBAUM: The term tropical ulcer covers a variety of conditions, but certain of them are supposed to be fusospirillary symbiotic infections. I object to the term gangrene for this particular type of ulcer and prefer the term phagedena. Andrews' textbook (Andrews, G. C.: Diseases of the Skin, Philadelphia, W. B. Saunders Company, 1930) is the only American book on dermatology which uses the term phagedena (at least in the index), but phagedena is a perfectly good term to indicate a more or less rapidly spreading but always progressive ulceration, with molecular destruction but not with loss of tissue in mass, as one finds in gangrene.

DR. ERICH URBACH (by invitation): I suggest performing a dextrose tolerance test. I have published the account of a case of this type in which I could show definitely that there was a decrease in dextrose tolerance (Phagedänisches Geschwür der Brusthaut auf Grundlage eines Hautdiabetes, *Klin. Wchnschr.* **16**:1315 [Sept. 18] 1937), and after the administration of large doses of insulin I was able to cure this condition in three or four weeks, although I had tried all types of therapy unsuccessfully for nine months previously.

NOTE.—A dextrose tolerance test was performed the following week, and the result was found to be normal.

Parapsoriasis Lichenoides Chronica. Presented by DR. ERICH URBACH (by invitation).

K. S., a white woman aged 25, presents, over the entire cutaneous surface except the face, hands and feet, poorly defined, red-brown lesions, 5 to 15 mm. in diameter. Some of the lesions are distinctly elevated, and some are covered with cigaret-paper-like olive green scales. There is no adenopathy. The patient, a nervous woman with pronounced tics, has had chorea repeatedly since the age of 13, the last attack being in April 1939. She had been given fever therapy with typhoid vaccine and various preparations for relief of symptoms. Eight months ago a papular eruption appeared on the chest and breasts, spreading rapidly over nearly the whole body. There was no itching. There was a positive Chvostek sign. The Wassermann reaction of the blood was negative. No fungi were demonstrable microscopically or by culture. Histologic examination revealed no characteristic changes except a moderate perivascular infiltration. The patient has been given solution of potassium arsenite and ultraviolet irradiation.

DISCUSSION

DR. JOSEPH V. KLAUDER: Dr. Urbach remarked to me privately that in Europe a number of these cases are observed but that here there are comparatively few.

DR. ERICH URBACH (by invitation): In Vienna I used to see patients with this condition in the clinics about once a month, but this is the first or second case I have observed in the two years I have been in this country. I do not know the reason for this. At present the condition is not Habermann's disease, although there may have been some resemblance previously.

Book Reviews

An Introduction to Medical Mycology. By George M. Lewis and Mary E. Hopper. Price, $5.50, cloth. Pp. 315, with 71 illustrations. Chicago: Year Book Publishers, 1939.

This volume will be welcomed by mycologists, dermatologists and physicians in general as the first really practical book on medical mycology in the English language. It is divided into two parts. The first part deals with the clinical, theoretic and experimental aspects of fungous diseases, and the second, with laboratory methods.

After a brief historical review of the progress of medical mycology, the authors discuss the classification, structure and physiologic requirements of fungi. Fifteen methods for the diagnosis of fungous diseases are enumerated: clinical symptoms and signs, direct microscopic examination, cultures, filtered ultraviolet radiation, cutaneous tests, animal inoculation, fermentation, agglutination, precipitation and complement fixation tests, fusion of mycelia, histologic studies, therapeutic tests, autoinoculation and roentgenologic and spinal fluid examinations.

The chapter on immunity and cutaneous sensitization is based largely on the personal investigations of the authors. It gives a detailed review of the trichophytin test, its literature (American and European), technic, specificity and interpretation. The following and important points are made clear: 1. The trichophytin test has a considerable degree of specificity. 2. The reaction is not specific to a definite species or genus of fungi, so that a patient affected with one fungus will react to extracts made from different fungi and vice versa. 3. The presence of a fungous infection does not always produce sensitization to trichophytin. 4. Patients with allergic fungous eruptions (dermatophytid) almost always give a positive reaction, making the reaction, therefore, a requisite to a diagnosis of dermatophytid. 5. Repeated tests, even on the same area of the skin, do not cause the appearance of false positive reactions. 6. The patch test with trichophytin is much less sensitive and has less significance than the intracutaneous test.

The chapter on superficial and deep mycoses includes all the common forms and, in addition, torulosis (European blastomycosis), chromoblastomycosis, tinea imbricata, aspergillosis and paracoccidioidal granuloma. Clinical descriptions are supplemented by immunologic data, results of cutaneous tests, examinations with filtered ultraviolet rays and methods of treatment and prophylaxis. Wherever possible, the relation between the species of fungi causing the disease and the clinical manifestations, immunologic reactions, prognosis and treatment is clearly brought out. Allergic eruptions due to fungi (trychophytid and dermatophytid) and the importance of fungous diseases as compensable dermatoses are also discussed.

The second part of the book is devoted to the laboratory methods used in mycologic investigation. This subject is almost entirely lacking in the standard textbooks of dermatology, pathology and bacteriology. Microscopic examination, artefacts which may cause confusion, cultivation of fungi, microscopic structure of cultures and animal inoculations are discussed. Then follows the description of 35 pathogenic fungi in regard to their clinical characteristics, immunologic reactions, microscopic structure in the lesions, gross and microscopic structure of cultures, examination with filtered ultraviolet rays, animal inoculations and differential diagnosis. The book closes with the description of 15 species of fungi which occur as common contaminants. These should be familiar to a mycologist in order to avoid mistaking them for pathogenic types.

In a brief review it is impossible to mention all the topics treated in this book, which is full of practical observations and instructions which will be of great value to physicians and laboratory workers engaged in mycologic investigation.

Among mycologists certain points at variance with the opinions presented in the literature may cause discussion. For instance, in dividing Trichophyton into endothrix and ectothrix fungi the authors define the term ectothrix as a fungus "which forms chains of spores external to the hair" (p. 5). However, this term is taken by many mycologists to mean a fungus which grows inside and outside the hair. In the description of tinea capitis due to Trichophyton crateriforme, it is said that the "infected hairs do not break off at the surface of the scalp" (p. 50). The reader may therefore be under the impression that this fungus does not produce broken-off hairs. As a matter of fact, hairs of the scalp infected with T. crateriforme do break off, but are 2 to 3 mm. long, somewhat longer than those in cases due to Trichophyton violaceum or Trichophyton acuminatum. In the latter cases the hairs are short, broken and twisted on the surface of the scalp in the form of comedo-like black dots.

The suggestion to abolish the genus Achorion and include fungi usually placed in this genus (for instance, Achorion schönleinii) under the genus Trichophyton endothrix (pp. 5 and 50) may meet with objections. The clinical peculiarities of favus differ distinctly from what is observed in trichophyton infections. The formation of large colonies (scutula) in the lesions is a biologic property of the fungus Achorion which is fundamentally different from that of all other pathogenic fungi. The tendency of the common human species, A. schönleinii, to persist in the scalp for decades causing atrophy and permanent alopecia is also a biologic feature not present in T. endothrix. The histologic structure of a scutulum is pathognomonic. The microscopic picture of a favic hair in a potassium hydroxide preparation, with the cylindric air spaces inside the hair and round air bubbles on the surface, is also characteristic. Cultures of A. schönleinii present a peculiar combination of a polymorphous mycelium, with terminal knoblike swellings, hornlike and candle-stick like branchings, pectinate bodies, protoplasmic masses bursting through the membrane of the hyphae, chlamydospores, microconidia and rudimentary fuseaux. All these clinical, microscopic and cultural features justify the separation of the genus Achorion from the genus Trichophyton. It is also worth noting that A. schönleinii grows not only inside the hair but also outside of it, forming a dense mass of filaments and spores around the hair in its intrafollicular portion. In view of the foregoing observations, if the genus Achorion must lose its independence to be annexed by its neighbor Trichophyton, it would be more justifiable to consider Achorion an ectothrix rather than an endothrix.

Some contradictions can be found in the description of the microscopic structure of cultures of Achorion. It is said (p. 227, line 6 above), "pectinate bodies not seen in . . . Achorion," while on the same page (line 3 below) pectinate bodies are mentioned as a feature of the genus Achorion. On page 227 it is said that the genus Achorion produces "no fuseaux, no nodular organs" (lines 1 and 2 below), but on page 292 it is stated that in the cultures of the species Achorion quinckeanum "fuseaux and nodular organs may be seen."

The authors considered Trichophyton purpureum, Trichophyton interdigitale, Trichophyton niveum and Epidermophyton inguinale all variants of Trichophyton gypseum (p. 259). Many authorities have described these five fungi as distinct species because of differences in their relations to the hairs, in the gross and microscopic structure of their cultures and in their pathogenicity for animals.

Trichophyton rosaceum is mentioned briefly as an endothrix. Sabouraud and others call it an ectothrix, because it grows in abundance not only inside but also outside of hairs.

All these differences of opinion enhance the value and interest of the book. It is not merely a compilation of the literature but a product of years of personal observation and independent thinking.

The external appearance of the book and its typographic qualities leave nothing to be desired. The paper is excellent, and the print, large and clear. Seventy-one beautiful plates give over three hundred and fifty illustrations, presenting clinical pictures of mycoses, the microscopic structure of fungi in cutaneous material, the gross and microscopic structure of cultures, and histologic sections. The abundance of illustrative material makes the book a veritable atlas of medical mycology and facilitates greatly the study of this eminently descriptive branch of science.

Dermatological Allergy. By Marion B. Sulzberger, M.D. Price, $8.50. Pp. 518, with 17 tables, 39 black and white illustrations and 13 color illustrations. Springfield, Ill.: Charles C. Thomas, Publisher, 1940.

This book of Sulzberger's is excellent. A close and enthusiastic student of allergy, a persistent investigator in the field, he has put on paper in lecture form a well thought-out presentation of the story of allergy, using dermatologic phraseology and clinicodermatologic concepts to bring allergy and its implications to the doorstep of the dermatologist. In sixteen chapters (lectures), beginning with "On Definitions and Classifications" and closing with "On the Future of Allergy," is given a complete, though selected, picture of the allergist at work in the elaboration of an understanding of clinical dermatology. Tables and appendixes are effectively employed to assist in the capitulation and understanding of the text, which is further augmented by thirty-nine black and white illustrations and thirteen colored plates.

This unqualified encomium on the substance of this book cannot be carried over to its form. The reviewer wishes there were less italics and less small print and, further, hopes that the next edition will find the "lecture" form completely discarded and the presentation made in the third person.

The author shows rare courage in putting on paper his last lecture, "On the Future of Allergy." Surely this must be subject to change without notice. Certainly the immediate future of allergy in medicine will be shaped in greatest measure by the influence on it of experience with and understanding of the latest —and spectacular—recrudescence of chemotherapy.

This book is unhesitatingly recommended to student and practitioner, and it deserves a place in every dermatologist's library.

News and Comment

DEATH NOTICE

Kenneth Mercereau Davenport, aged 38, Watertown, N. Y., a passed assistant surgeon in the United States Public Health Service reserve and a member of the American Academy of Dermatology and Syphilology, died on February 15, of burns received when he attempted to rescue his family from a burning building.

INDEX TO VOLUME 41

The asterisk (*) preceding the page number indicates an original article in the Archives. Subject entries are made for all articles. Author entries are made for original articles and society transactions. Book Reviews, Obituaries and Society Transactions are indexed under these headings in their alphabetical order under the letters B, O and S, respectively.

Abnormalities and Deformities: See under names of diseases, organs and regions, as Atrophy; Ectodermal Defect; Fingers and Toes, abnormalities; Hair, abnormalities; Nails; Pigmentation; Skin, abnormalities; etc.

Abramowitz, E. W.: Basal cell epithelioma? 614
Erythroderma psoriaticum cured with 30 per cent sulfur in petrolatum; coincident chronic lymphatic leukemia and recurrent herpes zoster, 136
Fixed eruption from magnesium hydroxide; polysensitivity, *707
Leukaemia cutis, 941

Abscess: See under names of organs and regions, as Skin; etc.

Acanthosis nigricans, 797
rubra lichenoides, autohemotherapy for, 742
rubra lichenoides, autohemotherapy for; diagnostic comments on article by Antonio Risi, 742

Acarophobia: See under Scabies

Achondroplasia, scleroderma in chondrodystrophic person, 596

Achromia: See Nevi; Pigmentation, cutaneous

Acid, Ascorbic: See Vitamins, C
Cevitamic: See Vitamins, C
Nicotinic: See also under Pellagra; Stomatitis; etc.
nicotinic, dermatitis from industrial contact with, 130
Tannic: See Burns

Acne and tuberculosis, 936
conglobata, 1150
new acne stilet, *1108
new observations on iritis rosacea; report of case, 578
rosacea; dermatologic aspect of affections of eye, 1114
rosacea, treatment with extract of cortex of adrenal gland, 578
rosacea with keratitis and corneal ulceration, 184
sensitization of skin induced by turbinal pollen application, resulting in facial acne, 1114
unilateral acneform eruption (following use of cold cream), 795
urticata polycythaemica, 807
varioliformis, 1135
varioliformis treated with bacteriophage, 969
vitamin D in treatment, *835
vitamin therapy in dermatology with reference to vitamin D in treatment of acne and of diseases due to altered usage of calcium, *842
vulgaris; blood iodine of patients with acne vulgaris, *506
vulgaris, therapy with modified liver extract; further studies, 932

Acrocyanosis: See Cyanosis

Acrodermatitis chronica atrophicans, 750
chronica atrophicans and senile elastosis, 168

Acrodynia, malignant type, 388

Acrosclerosis: See Scleroderma

Actinodermatitis: See under Roentgen Rays

Actinomyces, superficial noninflammatory lesions of feet; demonstration in sections of fungus of genus Actinomyces, *1023

Actinomycosis, 411
pedis, case of, 577
sulfanilamide therapy in, 126

Addison's Disease, generalized lupus erythematosus, vitamin deficiency (pseudopellagra) or dermatomyositis, 185
lupus erythematosus involving lips, 771

Adenopathy: See Lymph Nodes

Adipose Tissue: See Fat

Adrenal Preparations, treatment of rosacea with extract of cortex of adrenal gland, 578
unusual reactions to slow epinephrine, 1114

Age, Old: See Old Age
syphilis with reference to incidence in relationship to age groups and status of therapy at date of infection, 129

Ainhum, syphilis or periarteritis, 443

Alcohol, contact dermatitis from rubber and alcohol, 1125

Allergy: See Anaphylaxis and Allergy

Allington, H. V.: Localized atrophy of skin and subcutaneous tissue (hemiatrophia faciei? scleroderma?), 437
Thickening and exfoliation of mucous membrane of mouth; case for diagnosis, 439
Urticaria from cold, 439

Alopecia: See also Hair, removal
arsenical dermatitis with pigmentation and alopecia, 761
cicatrisata; pseudopelade, 428, 985
cicatrisata; pseudopelade (Brocq), 948
trichomycosis axillaris flava associated with alopecia prematura, alopecia areata (chin) and fragilitas crinium (eyebrows and upper lip), 449

Alveolar Process: See Jaws

Aminopyrine, bullous and pustular eruption of face due to, 936
multiple symmetric gangrene occurring during prolonged administration of aminopyrine; report of case, *678

Amshel, F.: Scleroderma or Bazin's disease, 951

Amyloidosis cutis, localized (lichenoid type), 605

Amyotrophy: See Atrophy, muscular

Anaphylaxis and Allergy: See also Food, allergy; and under names of various diseases, as Asthma; Eczema; Rhus, poisoning; etc.
allergy in childhood; its onset and natural progress, 125
delayed positive reaction to patch test with nupercaine ointment, 1141
desensitization in treatment of menstrual intoxication and other allergic symptoms, 130
list of substances for patch-testing and concentrations to be employed, 386
new modification of patch test (chamber method), *649
passive transfer of light sensitivity; report of case, *889
study of cutaneous tests of intolerance, 933
whealing capacity of skin of newborn or young infant; report of experiments, *1029

Ancylostomiasis, creeping eruption due to, 1139

Anderson, C. R.: Pachyonychia congenita; dermatitis of hands, 772

Anderson, N. P.: Fixed arsenical pigmentation, 442
Parapsoriasis (en plaque type)? 441

Andrews, G. C.: Colloid milium, 788
Congenital defects of skin, eye and bones, 135
Iododerma, contact dermatitis or dermatitis medicamentosa, 957
Lupus erythematosus and lipogranulomatosis, 965
Psoriasis and granuloma annulare, 133
Androgens, androgenic substance and sweat, *654
influence of ultraviolet irradiation on excretion of sex hormones in male, 386
urinary excretion of estrogenic substances after administration of testosterone propionate, 386
Anemia, pernicious, with mycosis fungoides, 616
Anesthesia, dermatitis due to butyn, 126
Anetoderma: See Atrophy
Aneurysm, aortitis; aortic regurgitation; aneurysm of thoracic aorta, 162
tertiary syphilis; aneurysm of ascending portion of aorta, 161
Angiokeratoma corporis diffusum, first case in Japan, 743
Angioma serpiginosum, 609, 751
Anhidrosis: See Sweat Glands
Anthrax, outbreak in minks with infection of ranch owner, 127
Antigens and Antibodies, cutaneous absorption; direct technic for demonstrating percutaneous absorption of antigens, *692
Anus: See also Rectum
herpes zoster analis, 743
Pruritus: See Pruritus
Aorta, aortitis; aortic regurgitation; aneurysm of thoracic aorta, 162
Aortitis; aortic regurgitation; aneurysm of thoracic aorta, 162
Aphthae: See Stomatitis
Apparatus: See also Instruments
absorption of roentgen rays by skin; experimental determinations, *87
natural color photography of skin; new apparatus for proper illumination, *527
qualitative measurements of low voltage shockproof x-ray tubes, *718
Appel, B.: Lupus vulgaris, 427
Myositis ossificans, 410
Argyria: See under Silver
Armenteros, J. A.: Pinta in Cuba; special clinical features of Cuban cases and discovery of spirochete in active lesions and in lymph glands, *463
Arrhenoblastoma, case which simulated Cushing's disease, 385
Arsenic and Arsenic Compounds: See also Arsphenamines; Tryparsamide
action of sulfur-containing compounds in arsenical and mercurial poisoning with review of use of sodium thiosulfate in dermatologic practice and report of experiments on rabbits with sodium thiosulfate, sodium p-sulfhydril phenyl sulfonate and sodium formaldehyde sulfoxylate, *308
arsenical dermatitis (chronic), 590
arsenical dermatitis with pigmentation and alopecia, 761
arsenical keratosis; case for diagnosis, 962
arsenical pigmentation and keratoses, 431
arsenical pigmentation and keratoses first manifested in patient at age of 6 years, 778
arsenical pigmentation and keratosis of palms and soles in patient with chronic myelogenous leukemia, 802
complications due to arsenical therapy in syphilitic pregnant women; report of 7 maternal deaths, 128
death following mapharsen therapy, 384
fixed arsenical pigmentation, 442
fixed eruption due to mapharsen, 943
fixed eruption with extracutaneous manifestation due to mapharsen; report of case, *509
Arsenic and Arsenic Compounds—Continued
herpes zoster occurring during treatment for syphilis, *929
Therapy: See under Syphilis; etc.
Arsphenamines, caution in arsphenamine therapy (report of case of purpura haemorrhagica following treatment with neoarsphenamine), 1111
influence of sodium dehydrocholate on therapeutic activity of neoarsphenamine; experiments with acute testicular syphilis of rabbits, *838
overtreatment in syphilis, 1112
postarsphenamine jaundice, 166
postarsphenamine jaundice; treatment of, 383
role of sympathetic nervous system in reactions to arsenical chemotherapy, 740
simplified technic for administering old arsphenamine, 384
Therapy: See under Syphilis
Arthritis, acute, associated with myocarditis simulating acute lupus erythematosus; report of case, *1053
atrophic, with subcutaneous nodules and pulmonary tuberculosis, 144
atrophic, with subcutaneous nodules and tertiary syphilis, 143
deformans; relief of arthritic symptoms following urticaria, 1113
multiple symmetric gangrene occurring during prolonged administration of aminopyrine; report of case, *678
Aschoff-Klinge Nodule: See Rheumatic Fever
Asthma: See also Anaphylaxis and Allergy
unusual reactions to slow epinephrine, 1114
Astrachan, G. D.: Syphilis or carcinoma of tongue, 979
Atrophoderma: See Atrophy
Atrophy: See also Acrodermatitis chronica atrophicans; Poikiloderma atrophicans vasculare; and under names of organs and regions, as Face; Nerves, optic; Penis; etc.
atrophia cutis maculosa, case for diagnosis, 415
localized, of skin and subcutaneous tissue (hemiatrophia faciei? scleroderma?), 437
macular, nonsyphilitic white spot disease or anetoderma erythematodes of Jadassohn, 147
muscular, with meningomyelitis syphilitica, 158
pseudoatrophoderma colli (?), 430
syphilitic macular atrophy, 151
tertiary syphilis and syphilitic macular atrophy, 811
Autohemotherapy: See Serotherapy and Hemotherapy
Avakian, E. V.: Relapsing febrile nonsuppurative panniculitis; report of 2 cases, *681
Avitaminosis: See under Vitamins
Axilla: See Sweat Glands

Bacteria: See Staphylococci; Streptococci; Viruses; etc.
Bacterid or psoriasis (pustular), 580
psoriasis or dermatophytosis with dermatophytide? 1140
pustular, 140
Bacteriophage: See under Acne
Baer, R. L.: Whealing capacity of skin of newborn or young infant; report of experiments, *1029
Bailey, H.: Cutaneous and subcutaneous calcinosis; scleroderma (right cheek)? 1140
Eczema vaccinatum, 1137
Leukoderma following dress shield dermatitis, 1141
Balanitis: See under Penis
Baldness: See Alopecia
Bancroft, I.: Herpes zoster occurring during treatment for syphilis, *929
Barker, L. P.: Tertiary syphilis; aneurysm of ascending portion of aorta, 161
Barnes, B.: Androgenic substance and sweat, *654

Bazin's Disease: See Erythema induratum
Bechet, P. E.: Acne varioliformis treated with bacteriophage, 969
Atypical erythema induratum, case for diagnosis, 135
Neurotic excoriations, case for diagnosis, 966
Periphlebitis nodularis necrotisans; attempt at definition and classification, *55
Report on case of ulcer of tongue, 798
Tattoo marks; result of attempts to remove them, 619
Becker, S. W.: Action of sulfur-containing compounds in arsenical and mercurial poisoning with review of use of sodium thiosulfate in dermatologic practice and report of experiments on rabbits with sodium thiosulfate, sodium p-sulfhydril phenyl sulfonate and sodium formaldehyde sulfoxylate, *308
Bullous disease; bullous Darier's disease (Pels and Goodman); familial benign pemphigus (Hailey and Hailey); herpetiform dermatitis repens (Ayers and Anderson), 1170
Histiocytoma, case for diagnosis, 1151
Lichen planus of eyelids, case for diagnosis, 1169
Parakeratosis variegata (lymphoblastoma?), 603
Pityriasis rubra pilaris, 599
Poikiloderma atrophicans vasculare, dermatomycosis and onychomycosis, 581
Prurigo of Hebra or pigmentation? 600
Tuberculosis verrucosa cutis, 779
Behrman, H. T.: Recklinghausen's disease; its elusive manifestations and internal relations, *480
Beinhauer, L. G.: Cheilitis and dermatitis from tooth paste, *892
Bell's Palsy: See Paralysis, facial
Benedek, T.: Reticuloendotheliosis, case for diagnosis, 780
Berkovsky, M.: Parapsoriasis varioliformis acuta, 976
Berlin, C.: Leishmaniasis recidiva cutis; leishmanid, *874
Berman, S.: Acute syphilitic transverse myelitis; clinical study and report of case, *1078
Bernstein, E. T.: Exudative discoid and lichenoid chronic dermatosis (phase resembling leukotic dyscrasia), 1185
Besnier-Boeck's Disease: See Sarcoid
Bingham, C. T.: Reticulum cell sarcoma; report of case with pronounced cutaneous manifestations, *722
Binkley, G. W.: Pustular bacterid, 140
Biopsy: See under Bones, marrow; etc.
Bismuth and Bismuth Compounds, effect of adding chlorobutanol to bismuth subsalicylate in oil injected intramuscularly, *380
effect of bismuth therapy on latent plumbism; preliminary report, *38
erythema of ninth day following bismuth therapy for syphilis, *1076
erythroderma in course of bismuth therapy, 933
lichen planus or dermatitis medicamentosa from bismuth, 627
overtreatment in syphilis, 1112
perioral pigmentation due to bismuth? 966
tertiary syphilis; transverse myelitis following administration of bismuth compound, 160
Therapy: See under names of various diseases, i. e., Lupus erythematosus; etc.
Blastomycosis, 738. See also Coccidioides; Moniliasis
chromomycosis (dermatitis verrucosa) of face; report of case and study of causative organism, Phialophora verrucosa, *42
Blastomycosis—Continued
comparison of infection of mice by mycelial and yeast forms of Blastomyces dermatitidis, 739
systemic, 758
Bleier, A.: Erythema annulare rheumaticum (Lehndorff-Leiner); report of case, *515
Blood, Diseases: See Anemia; Leukemia; etc.
iodine of patients with acne vulgaris, *506
transfusions; cases of familial and of conjugal pemphigus vulgaris; use of transfusions of blood from cured patients, *1073
Bloom, D.: Hereditary ectodermal dysplasia of anhidrotic type associated with syndactylia and lichen nitidus, 946
Hereditary trophedema (Milroy-Meige disease) of extremities associated with ptosis palpebrae in 3 generations, 611, 939
Leukoderma acquisatum centrifigum (Sutton), 771
Boardman, W. P.: Erythema multiforme, 425
Lichenoid dermatitis? 421
Lupus erythematosus or Besnier-Boeck-Schaumann disease, 416
Tertiary syphilodermas, 426
Boeck Sarcoid: See Sarcoid
Boerner, F.: Simplified complement fixation technic for diagnosis and treatment of syphilis; its sensitivity and specificity, *32
Bones: See also under names of bones
congenital defects of skin, eye and bones, 135
Diseases: See Osteitis; Osteomyelitis; etc.
marrow; limitations of biopsy of sternal marrow, 385
tuberculosis; osteitis tuberculosa multiple cystoides and sarcoid lesions, 574
Book Reviews:
Argyria: Pharmacology of Silver; W. E. Hill and D. M. Pillsbury, 995
Dermatological Allergy; M. B. Sulzberger, 1200
Directory of Medical Specialists Certified by American Boards, 1939; P. Titus and J. S. Rodman, 995
Introduction to Medical Mycology; G. M. Lewis and M. E. Hopper, 1198
Manual of Dermatology; C. S. Wright, 996
Merck Index, 188
Text-Book of Occupational Diseases of Skin; L. Schwartz and L. Tulipan, 633
Tumors of Skin, Benign and Malignant; J. J. Eller, 633
Bordet-Wassermann Reaction: See under Syphilis
Bourneville-Pringle's Disease: See Sclerosis, tuberous
Bowen's Disease: See Cancer, precancer
Brain: See also Medulla Oblongata; Nervous System; etc.
Syphilis: See under Syphilis
tumors; coccidioidal granuloma simulating brain tumor in child of 4 years, 128
Breakstone, R.: Basal cell epithelioma located over sebaceous cyst, 978
Breast, chancre of both nipples, 976
Brocq's Disease: See Alopecia cicatrisata
Brown, H.: Prevention of industrial dermatitis with reference to protective hand creams, soap and harmful role of some cleansing agents, *331
Brunsting, L. A.: Acrodermatitis chronica atrophicans, 750
Angioma serpiginosum, case for diagnosis, 751
Dermatitis herpetiformis, 755
Generalized erythroderma (Hodgkin's disease?), 753
Group of cases of lupus erythematosus, 745
Group of cases of pyoderma with various systemic manifestations, 752
Hodgkin's disease, cutaneous and glandular, 756

Brunsting, L. A.—Continued
Localized and linear scleroderma, atrophic and indurated, 749
Lupus vulgaris, 744
Perforating gumma of soft palate and nasal septum; asymptomatic neurosyphilis, 751
Rosacea-like tuberculid of Lewandowsky, 744
Sarcoid or lupus erythematosus, 760
Seborrheic eczema, 754
Senile pruritus (neurodermatitis?); hemangioendothelioma of left foot (Kaposi's sarcoma?), 754
Tinea capitis; kerion celsi, 748
Tinea capitis; kerion celsi; trichophytid, 748
Bubo, Venereal: See Chancroid
Buchanan, J. A.: Multiple symmetric gangrene occurring during prolonged administration of aminopyrine; report of case, *678
Buda, A. M.: Treatment of pruritus ani by tattooing with mercury sulfide, *521
Burgess, J. F.: Atopic dermatitis, 397
Extensive keloid following lightning, 392
Hypodermic sarcoid, 401
Lingua nigra, 390
Radiodermatitis; cellular keloid? 407
Recurrent vesicopustular eruption on extremities, 394
Tertiary syphilis, 392
Burns, cement burn; its etiology, pathology and treatment, 128
Buschke's Disease: See Scleroderma
Butterworth, T.: Balanitis xerotica obliterans, 629
Darier's disease in woman, 628
Epithelioma of penis, 629
Pemphigus of long duration in mouth, with acute exacerbation on skin; improvement following large doses of vitamin D, 630
Butyn: See under Anesthesia

Calcification; calcinosis cutis, 625
cutaneous and subcutaneous calcinosis; scleroderma (right cheek)? 1140
metabolic cutaneous; report of case with scleroderma, *864
metastatic; report of 2 cases, 385
Calcinosis: See Calcification
Calcium and Calcium Compounds, vitamin therapy in dermatology with reference to vitamin D in treatment of acne and of diseases due to altered usage of calcium, *842

Callaway, J. L.: Passive transfer of light sensitivity; report of case, *889
Camphor, acarophobia and tumors due to camphor in oil, 601
Cancer: See also Sarcoma; Tumors; and under names of organs and regions, as Eyes; Palate; Prostate; Tongue; etc.
carcinoma cutis, 1110
"common mole"; its clinicopathologic relations and question of malignant degeneration, *214
cutaneous metastatic carcinoma originating from osteomyelitic cavities, *545
manner of growth of frog carcinoma, studied by direct microscopic examination of living intraocular transplants, 574
metastases of scalp simulating turban tumors, *639
mortality from cancer of skin in relation to mortality from cancer of other sites; analysis of occupational mortality statistics of England and Wales, 1110
multiple epitheliomas; ulceration following use of "cancer paste," 1174
precancer; Bowen's disease of penis, 442
precancer; Bowen's disease or superficial epithelioma, 173
precancer; leukoplakia and allied mouth conditions, 1111
precancer; squamous carcinoma (Bowen's disease?), 1117
Cannon, A. B.: Arsenical keratosis, case for diagnosis, 962
Granuloma annulare or papulonecrotic tuberculid, 961
Tertiary syphilis; transverse myelitis following administration of bismuth compound, 160
Cantor, M. J.: Moniliasis, case for diagnosis, 176
Capillaries, capillaritis, 144, 803
Carbamide, Therapy: See under Verruca
Carbohydrates, cutaneous; normal skin, *193
Carbuncle, Malignant: See Anthrax
Carcinoma: See Cancer
Cardiovascular Diseases, necropsy survey of cardiovascular syphilis with reference to its decreasing incidence, 383
Carpenter, C. C.: Winter eczema, *569
Cataract, atopic eczema with bilateral cataract, 402
dermatogenous, 577
Cellulitis, recurrent, resulting in chronic lymphedema, 171
Cement burn; its etiology, pathology and treatment, 128
Cerebrospinal Fluid, occipital puncture in hands of dermatologist, 577
Chambers, S. O.: Ainhum, syphilis or periarteritis, 443
Chancre of both nipples, 976
Soft: See Chancroid
Chancroid, sulfanilamide in chancroid disease, 384
Charcot's Joints: See Tabes Dorsalis
Chargin, L.: Aortitis; aortic regurgitation; aneurysm of thoracic aorta, 162
Incipient granuloma inguinale, 806
Meningomyelitis syphilitica with muscular atrophy, 158
Syphilitic leukoderma following secondary papular eruption, 814
Chediak Reaction: See under Syphilis
Cheese, mite dermatitis, *831
Cheilitis: See under Lips
Chemotherapy: See under Arsphenamines
Chlorobenzene, dermatitis from orthodichlorobenzene, 128
Chlorobutanol, effect of adding chlorobutanol to bismuth subsalicylate in oil injected intramuscularly, *380
Chondrodystrophia: See Achondroplasia
Chorioretinitis: See Retinochoroiditis
Christian-Schüller Syndrome: See Schüller-Christian Syndrome
Christian-Weber's Disease: See Panniculitis
Chromidrosis: See under Sweat Glands
Chromomycosis: See Blastomycosis
Chrysocyanosis: See Tuberculosis, gold therapy
Cicatrix, scar of oriental sore, 413
Ciliary Body: See Uvea
Cinchophen and Cinchophen Derivatives, purpura due to ingestion of cinchophen, 1176
relief of arthritic symptoms following urticaria, 1113
Cincinnati Society of Dermatology and Syphilology, treatment of psoriasis with concentrated viosterol under auspices of, *664
Cinnabar: See under Mercury
Cipollaro, A. C.: Absorption of roentgen rays by skin; experimental determinations, *87
Erythema multiforme, case for diagnosis, 940
Mycosis fungoides with pernicious anemia? 616
Purpura annularis telangiectodes, 138
Purpura simplex, 612
Roentgen dosage in dermatology expressed in international roentgens, *1
Tertiary syphilis or moniliasis, 767
Xanthoma tuberosum multiplex, 617
Cisterna Magna, ambulatory suboccipital (cisternal) puncture, 743
Clarke, G. E.: Treatment of psoriasis with concentrated viosterol under auspices of Cincinnati Society of Dermatology and Syphilology, *664

Clarke, J. A., Jr.: Treatment of ragweed dermatitis; report of 7 cases of successful treatment with absolute alcohol extract of ragweed leaves, *551
Clavicle sign of late congenital syphilis; review of literature and report of 6 cases, *1060
Cleveland, D. E. H.: Mite dermatitis, *831
Clijat-Petges Disease: See Poikiloderma
Coccidioides; coccidioidal granuloma, 990
coccidioidal granuloma simulating brain tumor in child of 4 years, 128
Cohen, D. M.: Cutis hyperelastica, 1169
Pigmentation (fixed), 603
Cohen, J. A.: Lymphogranuloma venereum affecting simultaneously cervical and inguinal lymphatic glands; report of case, *557
Cold, allergy due to, 1149
urticaria from, 439
Cold Cream: See under Creams
Cole, H. N.: Angioma serpiginosum, 609
Dermatitis repens, 1176
Radiodermatitis of hands with malignant degeneration, 149
Urticaria pigmentosa, 798
Colitis, chronic ulcerative; erythema multiforme; infectious eczematoid dermatitis, 752
chronic ulcerative; pyoderma, 752
hidrosadenitis and pyoderma with colitis, 753
ulcerative; pyoderma gangraenosum treated with sulfanilamide; report of case, *711
Collagen, imbibitio lipoidica collageni degenerati cutis, 180, 1193
Colloid milium, 788
Cologne Water, photosensitizing agents; brief review of literature, 1112
Combes, F. C.: Blue nevus (Jadassohn), 959
Infectious eczematoid dermatitis, 768
Lupus vulgaris, 795
Perioral pigmentation due to bismuth? 966
Sarcoidosis, 793
Superinfection in syphilis; report of possible case, *1108
Unilateral acneform eruption (following use of cold cream), 795
Complement Fixation: See under Syphilis
Condom dermatitis, 1160
Congress: See Societies
Conjunctiva, lupus erythematosus of, 388
pemphigus of, 429
Conjunctivitis, erythema exsudativum multiforme (Hebra) with conjunctivitis and stomatitis, *1066
Coombs, F. S.: Pemphigus; experimental studies on 34 patients, *359
Copper, methods for controlling schistosome dermatitis (use of copper carbonate to kill snails), 1113
Copra, mite dematitis, *831
Cornbleet, T.: Androgenic substance and sweat, *654
Cutaneous carbohydrates; normal skin, *193
Cutis hyperelastica, 1169
Erythema multiforme or drug eruption, 786
Excretion of sulfanilamide through skin, *912
Hemolymphangioma (preponderant hemangioma transformed to lymphangioma), 785
Herpes zoster in infant 3 months old, 782
Hodgkin's disease, 783
Koilonychia, 784
Necrobiosis lipoidica, 594
Necrobiosis lipoidica diabeticorum, 594
Oil tumors, case for diagnosis, 1153
Pigmentation (fixed), 603
Psoriasis (pustular) or bacterid? 580
Sarcoid (Schaumann's syndrome), 591
Cornea, Inflammation: See Keratitis
rosacea with keratitis and corneal ulceration, 184
Corson, E. F.: Lichen sclerosus et atrophicus, 982
Cosmetics: See also Cologne Water; Lipstick
dangers in use of, *64
measurable physicochemical changes in hair as result of therapeutic and cosmetic applications, 742
protection of cosmetic consumer, *75
unilateral acneform eruption (following use of cold cream), 795
vitamins in, *74
Costello, M. J.: Dermatitis herpetiformis treated with sulfapyridine, 134
Fixed eruption due to mapharsen, 943
Kaposi's sarcoma and leukonychia, 618
Lupus erythematosus, 613, 618
Lupus erythematosus with tuberculin reaction, 942
Lymphogranuloma venereum affecting simultaneously cervical and inguinal lymphatic glands; report of case, *557
Minor congenital ectodermal cutaneous defect in naevus linearis distribution, 132
Pigmented purpuric lichenoid dermatitis, case for diagnosis, 945
Creams, facial; composition and dangers, *68
prevention of industrial dematitis with reference to protective hand creams, soap and harmful role of some cleansing agents, *331
unilateral acneform eruption following use of cold cream, 795
Creeping eruption due to ancylostomiasis, 1139
Cross, F. E.: Chancre of both nipples, 976
Csillag's Disease: See Dermatitis lichenoides chronica atrophicans
Cummer, C. L.: Onychia due to handling sugar, 142
Curtis, G. H.: Cutaneous hypersensitivity to triethanolamine; report of 2 cases, *729
Elephantiasis of lips and of male genitalia with reference to syphilis and lymphogranuloma venereum as etiologic factors, *11
Necrobiosis lipoidica diabeticorum, 151
Pityriasis lichenoides chronica, 150
Cushing's Disease: See Pituitary Body, tumors
Cutis Elastica: See Skin, abnormalities
Hyperelastica: See Skin, abnormalities
Cyanosis, temperature measurements in neighborhood of acrocyanosis and lupus vulgaris, 743
unilateral acrocyanosis, 172
Cysts: See under names of organs and regions, as Sebaceous Glands; etc.

Dactylitis: See Fingers and Toes
Dahr-Chediak Reaction: See under Syphilis
Dandruff: See Seborrhea
Danlos-Ehlers Syndrome: See Joints; Skin, abnormalities; Tumors
Darier's Disease: See Keratosis follicularis
Darier-Roussy Sarcoid: See Sarcoid
Decker, D. A.: Lichen nitidus, 626
Vitiligo with superimposed pellagra, 625
Deficiency Diseases: See Pellagra; Vitamins
Dementia Paralytica, juvenile; congenital syphilis; interstitial keratitis, 167
malarial treatment of general paresis; relation of height, duration and frequency of fever to clinical and serologic results, 384
nitritoid reaction to tryparsamide; report of case, *690
DeOreo, G. A.: Dermatitis factitia, 139
Livedo racemosa in syphilitic patient, 152
Localized amyloidosis cutis (lichenoid type), 605
Necrobiosis lipoidica diabeticorum, 152
Purpura due to ingestion of cinchophen, 1176
Depigmentation: See Pigmentation
Depilatories: See Hair, removal
Dermatitis: See also Acrodermatitis; Eczema; Neurodermatitis; etc.
Acneform: See Acne
and cheilitis from tooth paste, *892
Arsphenamine: See Arsphenamines
artefacta: localized bullous eruption with keloidal scarring, 632

Dermatitis—Continued
Atopic: See Eczema; Neurodermatitis
Atrophicans: See Atrophy
dangers in use of cosmetics, *64
due to butyn, 126
due to safety pins; interesting sequence of sensitization due to contact with nickel, *568
Eczematous: See Eczema
exfoliative, mycosis fungoides or tumors of face, 970
exfoliative; syphilis with negative serologic changes, 391
exfoliative, treated with sulfanilamide, 186
exudative discoid and lichenoid chronic dermatosis (phase resembling leukotic dyscrasia), 1185
factitia, 139
factitia; autophytic dermatitis, 1114
factitia; neurotic excoriations, case for diagnosis, 966
Gangraenosa: See Gangrene
generalized exfoliative, vitamin C in, *660
heat sensitivity (controlled by estrogen therapy), 1111
herpetiformis, 755, 1136
herpetiformis; bullous disease; bullous Darier's disease (Peis and Goodman); familial benign pemphigus (Hailey and Hailey); herpetiform dermatitis repens (Ayers and Anderson), 1170
herpetiformis, erythema multiforme or pemphigus, 444
herpetiformis; generalized Trichophyton purpureum infection simulating dermatitis herpetiformis; report of case, *732
herpetiformis or recurring erythema multiforme, 968
herpetiformis perstans, 773
herpetiformis; recurrent bullous eruption (Duhring's disease?) treated with trichophytin, 812
herpetiformis treated with sulfapyridine, 134
histamine-like effect of irradiated or inflammatory skin, 741
lichenoid, 421
lichenoides chronica atrophicans (Csillag) or lichen planus sclerosus et atrophicus, 422
Medicamentosa: See also under Drugs; and under names of drugs, as Arsenic and Arsenic Compounds; Arsphenamines; Bismuth and Bismuth Compounds; Gold and Gold Compounds; Phenolphthalein; Silver; Tryparsamide; etc.
medicamentosa; drug eruption, lichen planus or pityriasis lichenoides et varioliformis acuta, 765
medicamentosa, iododerma or contact dermatitis, 957
medicamentosa or Senear-Usher pemphigus, 795
mite dermatitis, *831
Multiformis: See Dermatitis herpetiformis
Occupational: See Industrial Diseases
pachyonychia congenita; dermatitis of hands, 772
pigmented purpuric lichenoid dermatitis? 945
repens, 1176
Rhus: See Rhus, poisoning
Roentgen Ray: See under Roentgen Rays
Schistosome: See Schistosomiasis
Seborrheic: See Seborrhea
spontaneous necrotic dermatitis (hemolytic streptococcus), 414
Solar: See Light, toxicity
treatment of ragweed dermatitis; report of 7 cases of successful treatment with absolute alcohol extract of ragweed leaves, *551
Venenata: See also under Dyes; Nickel; Rubber; etc.
venenata, case for diagnosis, 177
venenata; condom dermatitis, 1160
venenata; iododerma, contact dermatitis or dermatitis medicamentosa, 957
Dermatitis—Continued
venenata; leukoderma following dress shield dermatitis, 1141
Verrucosa: See Blastomycosis
with depigmentation (pellagra?), 1152
Dermatologic societies, directory of, 189, 635, 997
Dermatolysis: See Skin, abnormalities
Dermatomycosis: See Mycosis, cutaneous
Dermatomyositis, case for diagnosis, 597
generalized lupus erythematosus, vitamin deficiency (pseudopellagra), dermatomyositis or Addison's disease, 185
study of 40 cases, *1001
Dermatophytid: See Mycosis, cutaneous
Dermatophytosis: See Mycosis, cutaneous
Dermatoses: See Dermatitis; Eczema; Skin, diseases; etc.
Diabetes Mellitus: See also Xanthoma diabeticorum
hemochromatoses with diabetic bronzing, 798
lipodystrophy and lipomatosis from repeated injections of insulin, 389
necrobiosis lipoidica diabeticorum, 151, 152, 594, 757, 959, 1143
Diathermy: See under names of various diseases
Dickman, M.: Bowen's disease or superficial epithelioma? 173
Late secondary syphiloderm; syphilitic osteitis? 173
Dietrich, C.: Osteomatosis cutis, *562
Diphtheria, eczema of ear, associated with diphtheria-like organism; report of case, *1020
of vulva in children, 131
Directory of dermatologic societies, 189, 635, 997
Disinfection and Disinfectants, bacteriology of normal skin: new quantitative test applied to study of bacterial flora and disinfectant action of mechanical cleansing, 738
Dixon, R. S.: Beginning atrophy of optic nerves, 1123
Bilateral atrophy of optic nerves, 1122
Chorioretinitis, 1122
Eczema seborrhoeicum, with unusual persistent localization, 1121
Generalized scleroderma, 1119
Interstitial keratitis, 1122
Lupus erythematosus, 1117
Primary atrophy of optic nerve, 1122
Scleroderma, 1118, 1119
Sycosis vulgaris, 1116
Dorne, M.: Dermatomyositis, case for diagnosis, 597
Downing, J. G.: Dermatitis due to safety pins; interesting sequence of sensitization due to contact with nickel, *568
Nodular myxedema in case of thyrotoxicosis, 422
Dress shield; leukoderma following dress shield dermatitis, 1141
Driver, J. R.: Angioma serpiginosum, 609
Dermatitis repens, 1176
Radiodermatitis of hands with malignant degeneration, 149
Urticaria pigmentosa, 798
Drugs, Eruptions: See also Dermatitis medicamentosa; and under names of drugs, as Arsenic and Arsenic Compounds; Arsphenamines; Bismuth and Bismuth Compounds; Gold and Gold Compounds; Phenolphthalein; Tryparsamide; etc.
eruptions, or erythema multiforme, 786
Duhring's Disease: See Dermatitis herpetiformis
Durand-Nicolas-Favre's Disease: See Lymphogranuloma Venereum
Dyes, eruptions and photosensitivity due to dyed fabrics, *1044
eyelash dyes: composition and dangers, *73
hair dyes; composition and dangers, *71
Dyshidrosis: See Sweat Glands
Dystrophy, Adiposogenital: See Laurence-Biedl Syndrome

Ear, eczema of, associated with diphtheria-like organism; report of case, *1020
Ebert, M. H.: Fixed eruption, case for diagnosis, 1155
Lymphatic leukemia, 782
Melanoblastoma, 1167
Mycosis fungoides, 583
Psoriasis, case for diagnosis, 784
Psoriatic erythroderma, 1172, 1173
Pyoderma gangraenosum, 1156
Sarcoid of Boeck, 1154
Ecthyma, phagedenic, in infant, 389
Ectodermal Defect, congenital and familial hyperectodermosis, 933
hereditary ectodermal dysplasia of anhidrotic type associated with syndactylia and lichen nitidus, 946
minor congenital ectodermal cutaneous defect in naevus linearis distribution, 132
Eczema and its practical management, 125
atopic dermatitis, 397
atopic dermatitis or bacterial dermatitis, 417
atopic, with bilateral cataract, 402
depigmentation at site of chronic contact eczema, 621
dermatitis eczematosa (contact type) due to nickel, 815
dermatogenous cataract, 577
due to insulin, 933
erythema multiforme; infectious eczematoid dermatitis; chronic ulcerative colitis, 752
infectious eczematoid dermatitis, 768
infectious eczematoid dermatitis treated with sulfanilamide, 803
occupational, simulating knuckle pads (helioderma simplex annulare), 1148
of ear, associated with diphtheria-like organism; report of case, *1020
Seborrheic: See Seborrhea
urinary excretion of indoxyl (as indican) by infants with eczema, *113
vaccinatum, 1137
vitamin C in, *661
winter eczema, *569
Edema: See also Elephantiasis; and under names of organs
hereditary trophedema (Milroy-Meige) of extremities associated with ptosis of eyelids in several generations, 611, 939
lymphangiectasia of vulva with edema of legs, 146
Ehlers-Danlos Syndrome: See Joints; Skin, abnormalities; Tumors
Electrocoagulation: See under names of various diseases
Elephantiasis: See also Lymphogranuloma Venereum
of lips and of male genitalia with reference to syphilis and lymphogranuloma venereum as etiologic factors, *11
recurrent cellulitis resulting in chronic lymphedema, 171
Eller, J. J.: Eczema of ear, associated with diphtheria-like organism; report of case, *1020
Ellis, F. A.: Vitamin D in treatment of acne, *835
Emmet, R.: Blood iodine of patients with acne vulgaris, *506
Endocrine Glands: See also Hormones
Recklinghausen's disease; its elusive manifestations and internal relations, *480
Engeler, J. E.: Rosacea-like tuberculid of Lewandowsky? 1175
Syphilis (mucocutaneous relapse or reinfection?), 1177
Engman, M. F., Jr.: Inquiry into cause of pemphigus; is it virus disease? *78
Epidermolysis bullosa acquisita, 810, 1147
bullosa congenita, 434
bullosa hereditaria, 137
bullosa neonatorum, 993
bullosa or pemphigus, 434
bullosa with ichthyosis-like appearance; "epidermolysis bullosa et plicata," 936
Epidermomycosis; Epidermophytosis: See Moniliasis; Mycosis, cutaneous; Ringworm
Epilation: See Hair, removal
Epinephrine: See Adrenal Preparations
Epithelioma, atrophic process of glans penis with epithelioma, 432
basal cell epithelioma? 614
basal cell, located over sebaceous cyst, 978
basal cell, of glans penis? 781
calcified and mummified epitheliomas of skin in children, 388
erythroderma psoriaticum with tumor growths (epitheliomatous changes), 1180
fistulas from dead teeth simulating dermal epithelioma, *378
multiple benign epitheliomatosis, 774
multiple superficial epitheliomatosis, 174
multiple; ulceration following use of "cancer paste," 1174
of penis, 629
rate of ulceration of epitheliomas of skin and lip, *667
superficial, or Bowen's disease, 173
Epstein, E.: Bullous eruption following sulfanilamide therapy, *61
Effect of bismuth therapy on latent plumbism; preliminary report, *38
Epidermolysis bullosa (acquired), 1147
Eruptions and photosensitivity due to dyed fabrics, *1044
Pseudopelade, 428
Spiegler-Fendt sarcoid, 988
Tinea cruris and corporis, probably due to Trichophyton purpureum, 1146
Trichophyton purpureum infection, 989
Tryparsamide dermatitis, 428
Epstein, N N.: Argyria, 435
Case for diagnosis, 1146
Discrete inflammatory areas on vermilion border of lip; case for diagnosis, 991
Malignant melanoma, 433
Pemphigus or epidermolysis bullosa, 434
Recurrent erysipelas of face, 1145
Erb's Paralysis: See Paralysis, spinal
Eruptions: See under Arsenic and Arsenic Compounds; Arsphenamines; Drugs; Skin, diseases; etc.
Erysipelas, generalized dermatophytid after intracutaneous tests with trichophytin in dilutions of 1:30 and 2:0; combination of erysipelas and epidermophytosis, 983
recurrent, of face, 1145
sulfanilamide treatment, 127
Erysipeloid of Rosenbach successfully treated with sulfanilamide, *570
report of case, *1037
Erythema annulare rheumaticum (Lehndorff-Leiner); report of case, *515
arthriticum epidemicum, Haverhill fever; report of case with review of literature, 385
exsudativum multiforme giganteum, case of, 743
exsudativum multiforme (Hebra) with conjunctivitis and stomatitis, *1066
induratum, atypical, case for diagnosis, 135
induratum; scleroderma or Bazin's disease, 951
multiforme, 425, 940
multiforme; infectious eczematoid dermatitis; chronic ulcerative colitis, 752
multiforme or drug eruption, 786
multiforme, pemphigus or dermatitis herpetiformis, 444
multiforme, recurrent, of oral mucosa associated with progressive Fordyce's disease, 1192
multiforme, recurring, or dermatitis herpetiformis, 968
nodosum, rheumatic symptoms in, 937
of ninth day following bismuth therapy for syphilis, *1076
Erythema Dose: See Ultraviolet Rays
Erythroderma, generalized (Hodgkin's disease?), 753

Erythroderma—Continued
Ichthyosiforme: See Ichthyosis
in course of bismuth therapy, 933
psoriatic 1172, 1173
psoriaticum cured with 30 per cent sulfur in petrolatum; coincident chronic lymphatic leukemia and recurrent herpes zoster, 136
psoriaticum with tumor growths (epitheliomatous changes), 1180
Erythroplasia: See Penis
Esophagus, lesions in scleroderma, 934
Esthiomene: See Lymphogranuloma Venereum
Estrin, M. M.: Keratosis pilaris, 177
Lichen planus sclerosus et atrophicus (Hallopeau); case for diagnosis, 178
Estrogens: See also Hormones, sex
heat sensitivity (controlled by estrogen therapy), 1111
urinary excretion of estrogenic substances after administration of testosterone propionate, 386
Ethanolamine, cutaneous hypersensitivity to triethanolamine; report of 2 cases, *729
Ex-Lax: See Phenolphthalein
Extremities, Blood Supply: See also Raynaud's Disease
blood supply; intermittent venous compression in treatment of peripheral vascular disorders; report on 103 cases, 573
hereditary trophedema (Milroy-Meige) associated with ptosis of eyelids in several generations, 611, 939
Eyelash Dyes: See Dyes
Eyelids, dermatitis due to butyn, 126
dermatologic aspects of affections of eye, 1114
hereditary trophedema (Milroy-Meige) of extremities associated with ptosis of eyelids in several generations, 611, 939
lichen planus of, 1169
Eyes, cancer; manner of growth of frog carcinoma, studied by direct microscopic examination of living intraocular transplants, 574
congenital defects of skin, eye and bones, 135
Diseases: See Cataract; Glaucoma; etc.

Face, bullous and pustular eruption due to aminopyrine, 936
chromomycosis of; report of case and study of causative organism, Phialophora verrucosa, *42
hemiatrophy of, 147
hypertrophic nevus of scalp, face and oral mucosa, 1191
Paralysis: See Paralysis, facial
pyoderma faciale, *451
tumors, mycosis fungoides or exfoliative dermatitis, 970
Fat: See also Lipoids
lipodystrophy and lipomatosis from repeated injections of insulin, 389
Necrosis: See also Scleroderma
necrosis; subcutaneous adiponecrosis and lipogranulomatosis in infant, 576
necrosis; subcutaneous fat necrosis of newborn (obstetric lipophagic granuloma), 609
Favre-Nicolas' Disease: See Lymphogranuloma Venereum
Febricula: See Fever
Feer's Disease: See Acrodynia
Feigenbaum, H. L.: Multiple superficial epitheliomatosis, 174
Feiler, H. B.: Poikiloderma of Civatte, 809
Tuberculosis of skin, case for diagnosis, 977
Feldman, S.: Drug eruption, lichen planus or pityriasis lichenoides et varioliformis acuta, 765
Gumma of larynx, 979
Lymphogranuloma venereum, 977
Femur, osteoperiostitis of, 158
Fendt-Spiegler Sarcoid: See Sarcoid
Fever, Haverhill: See Erythema arthriticum epidemicum
spontaneous fever therapy in neurosyphilis, 127
Therapeutic: See under Syphilis; etc.
Fifth Venereal Disease: See Granuloma inguinale
Fingers and Toes: See also Foot
abnormalities; hereditary ectodermal dysplasia of anhidrotic type associated with syndactylia and lichen nitidus, 946
heloderma simplex et annulare (Vörner), 992
knuckle pads, 578
late secondary syphilis with pyogenic infection and syphilitic dactylitis, 449
Nails: See Nails
occupational eczema simulating knuckle pads (heloderma simplex annulare), 1148
osteomatosis cutis, *562
Fisher, A.: Lupus erythematosus, case for diagnosis, 947
Papular psoriasis, case for diagnosis, 949
Fistula from dead teeth simulating dermal epithelioma, *378
Flood, J. M.: Exfoliative dermatitis treated with sulfanilamide, 186
Fogo Salvagem: See under Pemphigus
Food, Allergy: See also under Anaphylaxis and Allergy; and under names of various foods
allergy; role of dietetics in dermatology including discussion of cutaneous manifestations of food allergy, 1112
Foot: See also Fingers and Toes; etc.
superficial noninflammatory lesions of feet; demonstration in sections of fungus of genus Actinomyces, *1023
Fordyce-Fox's Disease: See Fox-Fordyce's Disease
Foreskin: See Penis
Foster, P. D.: Pruritis ani and its relationship to seborrheic eczema and dermatophytosis, *699
Fourth Venereal Disease: See Lymphogranuloma Venereum
Fox, E. C.: Diseases of nails; report of cases of onycholysis, *98
Fox, H.: Lupus erythematosus disseminatus, 788
Mycosis fungoides (exfoliative dermatitis; tumors of face), 970
Parapsoriasis varioliformis acuta, 967
Recurring erythema multiforme or dermatitis herpetiformis, 968
Recurring lichen planus, 793
Fox-Fordyce's Disease, recurrent erythema multiforme of oral mucosa associated with progressive Fordyce's disease, 1192
Fragilitas Crinium: See Hair, abnormalities
Franks, A.: Arsenical dermatitis with pigmentation and alopecia, 761
Freeman, H. E.: Argyrosis, 606
Granuloma annulare; advanced bilateral cavitating pulmonary tuberculosis, 604
Keratosis blennorrhagia, case for diagnosis, 607
Lupus vulgaris of face; tuberculosis of buccal mucous membranes, pharynx and larynx; pulmonary tuberculosis, 1179
Lymphangioma and multiple hemangiomas in boy with adiposogenital dystrophy, 805
Melanosarcoma (cutaneous melanoblastoma), 141
Rosacea-like tuberculid of Lewandowsky; pulmonary tuberculosis, 608
Tuberculoma of palate and right angle of mouth; miliary pulmonary tuberculosis, 1179
Frei Reaction: See Lymphogranuloma Venereum
French, E. D.: Joseph Lee Kirby-Smith, 123
Friedlander, H. M.: Urticaria due to heat, 949

Friedman, R.: Erythroplasia of Queyrat, case for diagnosis, 1192
Recurrent erythema multiforme of oral mucosa associated with progressive Fordyce's disease, 1192
Reticulated pigmented poikiloderma of face and neck (Civatte), 631
Trichomycosis axillaris flava associated with alopecia prematura, alopecia areata (chin) and fragilitas crinium (eyebrows and upper lip), 449
Wood tick simulating pedunculated tumor, *887
Frost, K.: Bowen's disease of penis, 442
Fruchtbaum, L.: Schamberg's disease (unilateral), 170
Unilateral acrocyanosis, 172
Fuchs Reaction: See Syphilis
Fungi: See also Actinomycosis; Blastomycosis; Mycosis; Ringworm; Trichophytosis; etc.
chromomycosis of face; report of case and study of causative organism, Phialophora verrucosa, *42
cultural characteristics of Pityrosporum ovale; lipophylic fungus, 932
Malassezia furfur, cause of tinea versicolor; cultivation of organism and experimental production of disease, *253

Gangrene: See also Raynaud's Disease
multiple symmetric gangrene occurring during prolonged administration of aminopyrine; report of case, *678
Genitals: See under names of genitals, as Penis; Vagina; etc.
Gentian Violet: See Methylrosaniline
Germanin, Therapy: See Lupus erythematosus
Gilman, R. L.: Simplified complement fixation technic for diagnosis and treatment of syphilis; its sensitivity and specificity, *32
Glasses, nickel dermatitis from spectacle frames, 446
Glaucoma, recent periostitis developing during treatment of late syphilis, iridocyclitis (old) with secondary glaucoma, 157
Glomus Tumors: See under Tumors
Glossitis: See under Tongue
Goeckerman, W. H.: Lupus erythematosus, 774
Goiter, exophthalmic; nodular myxedema in case of thyrotoxicosis, 422
Gold and Gold Compounds, lichen planus following gold therapy, 763
pathogenesis of hyperkeratoses in gold dermatitis, 578
Therapy: See under Tuberculosis; etc.
Goldman, L.: Hypertherm treatment of cutaneous diseases, *919
Goldstein, M. B.: Sensitivity to gentian violet (methylrosaniline), *122
Goldstein's Disease: See Telangiectasis
Gonadotropic Substances: See Androgens; Hormones, sex
Gonococci, inguinal adenopathy of probable gonococcic origin, 932
Gotshalk, H. C.: Acute arthritis associated with myocarditis simulating acute lupus erythematosus; report of case, *1053
Relapsing febrile nonsuppurative panniculitis; report of 2 cases, *681
Gottron's Disease: See Ichthyosis
Graham, T. N.: Metabolic cutaneous calcinosis; report of case with scleroderma, *864
Granuloma: See also Lipogranulomatosis
annulare, 424
annulare; advanced bilateral cavitating pulmonary tuberculosis, 604
annulare and psoriasis, 133
annulare; eruption resembling granuloma annulare: case for diagnosis, 175
annulare of left leg, 792
annulare or papulonecrotic tuberculid, 961
Coccidioidal: See Coccidioides
Fungoides: See Mycosis fungoides
Granuloma—Continued
Inguinale, 1127. See also Lymphogranuloma Venereum
inguinale; facial paralysis due to early syphilis of central nervous system, 154
inguinale, incipient, 806
inguinale of vaginal wall and rectum with profuse hemorrhage, 801
Lymphadenoid: See Lymphocytoma
subcutaneous fat necrosis of newborn (obstetric lipophagic granuloma), 609
tuberculous, of nose, case for diagnosis, 435
Grau Triana, J.: Pinta in Cuba; special clinical features of Cuban cases and discovery of spirochete in active lesions and in lymph glands, *463
Graves, J. M.: Pemphigus vulgaris, 984
Syringomyelia (lumbosacral type), 433
Graves' Disease: See Goiter, exophthalmic
Greco, A. J.: Ainhum, syphilis or periarteritis, 443
Greenbaum, S. S.: Cases of familial and of conjugal pemphigus vulgaris; use of transfusions of blood from cured patients, *1073
Geometric phagedena (Brocq), 1195
Greenwood, A. M.: Atopic dermatitis or bacterial dermatitis, 417
Grenz Rays: See Roentgenotherapy
Grinnan, R. B.: Erysipeloid; report of case, *1037
Gross, E. R.: Distinctive type of tuberculosis in Negro associated with tuberculous keratouveitis, 445
Lupus erythematosus, 446
Nickel dermatitis from spectacle frames, 446
Prevention of industrial dermatitis with reference to protective hand creams, soap and harmful role of some cleansing agents, *331
Gross, P.: Poikilodermatomyositis (Peiges-Clijat), 1187
Grund, J. L.: Erythema of ninth day following bismuth therapy for syphilis, *1076
Guequierre, J. P.: Purpura annularis telangiectodes, 982
Gumma: See Syphilis, tertiary

Habermann's Disease: See Pityriasis lichenoides et varioliformis acuta
Hair, abnormalities; congenital fragilitas crinium, 791, 1134
color of; white forelock; new mutation, 575
Diseases: See also Alopecia
diseases; erythrokeratoderma of scalp and familial "trichorrhexis nodosa," 934
diseases; trichomycosis axillaris flava associated with alopecia prematura, alopecia areata (chin) and fragilitas crinium (eyebrows and upper lip), 449
Dyes: See Dyes
measurable physicochemical changes in hair as result of therapeutic and cosmetic applications, 742
removal; depilatories; composition and dangers, *74
Hallopeau's Disease: See Lichen planus sclerosis et atrophicus
Hand: See Fingers and Toes
Hand-Schüller-Christian Disease: See Schüller-Christian Syndrome
Hansen's Disease: See Leprosy
Hardy, M.: Late secondary syphilis with pyogenic infection and syphilitic dactylitis, 449
Primary atrophy of optic nerve; juvenile tabes dorsalis, 447
Hasley, C. K.: Beginning atrophy of optic nerves, 1123
Bilateral atrophy of optic nerves, 1122
Chorioretinitis, 1122
Eczema seborrhoicum, with unusual persistent localization, 1121
Generalized scleroderma, 1119
Interstitial keratitis, 1122

Hasley, C. K.—Continued
Lupus erythematosus, 1117
Primary atrophy of optic nerve, 1122
Scleroderma, 1118, 1119
Squamous carcinoma (Bowen's disease?), 1117
Sycosis vulgaris, 1116
Haverhill Fever: See Erythema arthriticum epidemicum
Hay Fever: See Anaphylaxis and Allergy; Pollen
Heat sensitivity (controlled by estrogen therapy), 1111
urticaria due to, 949, 1191
Hebra's Disease: See Prurigo
Heloderma simplex annulare; knuckle pads, 578
simplex annulare; occupational eczema simulating knuckle pads, 1148
simplex et annulare (Vörner), 992
Hemangioendothelioma, senile pruritus (neurodermatitis?); hemangioendothelioma of left foot (Kaposi's sarcoma?), 754
Hemangioma, hemolymphangioma (preponderant hemangioma transformed to lymphangioma), 785
lymphangioma and multiple hemangiomas in boy with adiposogenital dystrophy, 805
Hemiatrophy: See under Face
Hemochromatosis with diabetic bronzing, 798
Hemolymphangioma, preponderant hemangioma transformed to lymphangioma, 785
Hemotherapy: See Serotherapy and Hemotherapy
Herpes simplex; virus infection resembling herpes simplex, 179
zoster analis, 743
zoster and its visceral manifestations, 383
zoster and vitamin B_1, 1113
zoster; erythroderma psoriaticum cured with 30 per cent sulfur in petrolatum; coincident chronic lymphatic leukemia and recurrent herpes zoster, 136
zoster in infant 3 months old, 782
zoster occurring during treatment for syphilis, *929
Hetreed, F. W.: Necrobiosis lipoidica diabeticorum, 594
Hidrosadenitis: See under Sweat Glands
Hill, M. R.: Pruritus ani and its relationship to seborrheic eczema and dermatophytosis, *699
Hill, W. R.: Imbibitio lipoidica collageni degenerati cutis, 180
Histamine; histamine-like effect of irradiated or inflammatory skin, 741
Histiocytoma, case for diagnosis, 1151
Hodgkin's Disease: See Lymphogranuloma
Hoefer, H. F.: Effect of adding chlorobutanol to bismuth subsalicylate in oil injected intramuscularly, *380
Hollander, L.: Papular psoriasis, case for diagnosis, 949
Progressive pigmentary dermatosis, 952
Pseudopelade (Brocq), 948
Hopkins, J. G.: Actinic atrophy, case for diagnosis, 791
Chronic ulcer of back, case for diagnosis, 952
Poikilodermatomyositis, case for diagnosis, 790
Schamberg's disease, case for diagnosis, 789
Hopper, M. E.: Cultural variations of Trichophyton purpureum (Bang) with discussion of recognizable features, *895
Hormones: See also Adrenal Preparations; Androgens; Endocrine Glands; Estrogens; Insulin; etc.
sex; clinical evaluation of ovarian responses to gonadotropic therapy, 573
sex, significance in tanning of skin of women, 739
Humerus, lupus vulgaris; ulcers of tongue (unknown cause); osteomyelitis of right humerus, 624
Hydroa aestivale, 432
Hyperhidrosis: See Sweat Glands
Hyperkeratosis: See Ichthyosis; Keratosis
Hypernephroma with cutaneous metastases, 576
Hyperpyrexia: See under Syphilis
Hypophysis: See Pituitary Body
Hypothyroidism: See Myxedema

Ichthyosis, bullosa with ichthyosis-like appearance; "epidermolysis bullosa et plicata," 936
erythroderma progressivum symmetricum (Gottron) of congenital origin, 577
Icterus: See Jaundice
Imbibitio lipoidica collageni degenerati cutis, 180, 1193
Indican: See under Urine
Indoxyl in Urine: See Urine
Industrial Diseases, cement burn; its etiology, pathology and treatment, 128
dermatitis from industrial contact with nicotinic acid, 130
dermatitis from orthodichlorobenzene, 128
effect of bismuth therapy on latent plumbism; preliminary report, *38
leukoderma (occupational), 587
mortality from cancer of skin in relation to mortality from cancer of other sites; analysis of occupational mortality statistics of England and Wales, 1110
occupational dermatitis due to mint; report of 2 cases, *503
occupational eczema simulating knuckle pads (heloderma simplex annulare), 1148
onychia due to handling sugar, 142
prevention of industrial dermatitis with reference to protective hand creams, soap and harmful role of some cleansing agents, *331
Infants, newborn; subcutaneous fat necrosis of newborn (obstetric lipophagic granuloma), 609
newborn; whealing capacity of skin of newborn or young infant; report of experiments, *1029
Infectious Diseases: See Diphtheria; etc.
Ingels, A. E.: Hereditary hemorrhagic telangiectasia, 986
Lupus erythematosus, 436
Occupational eczema simulating knuckle pads (heloderma simplex annulare), 1148
Subacute lupus erythematosus, 987
Inguinal Glands: See Lymph Nodes
Injections: See also Blood transfusions
intradermal, actual site in skin, 126
Instruments: See also Apparatus
new acne stilet, *1108
Insulin: See also Diabetes Mellitus
eczema due to, 933
lipodystrophy and lipomatosis from repeated injections of insulin, 389
Iodine and Iodine Compounds, iododerma, contact dermatitis or dermatitis medicamentosa, 957
Iodobismitol: See Bismuth and Bismuth Compounds; Syphilis
Iododerma: See under Iodine and Iodine Compounds
Iridocyclitis, recent periostitis developing during treatment of late syphilis; iridocyclitis (old) with secondary glaucoma, 157
Iris, inflammation; new observations on iritis rosacea; report of case, 578
Iritis: See Iris, inflammation
Isaak, L.: Effective treatment of varicose ulcers of leg, *530
Ivy Poisoning: See Rhus, poisoning

Jadassohn's Disease: See Atrophy
Jaundice, chronic ulcer of leg associated with congenital hemolytic jaundice, 128
hemolytic, with ulceration of skin, 408
postarsphenamine, 166
postarsphenamine, treatment of, 383
Jaws, carcinoma of palate and alveolar process, 1168

John, M. B.: Treatment of ringworm of scalp with gentian violet, *370
Johnson, H. H.: Contact dermatitis due to physostigmine salicylate, 147
Granuloma inguinale of vaginal wall and rectum with profuse hemorrhage, 801
Hemochromatoses with diabetic bronzing, 798
Keratoderma palmare et plantare hereditarium punctatum, 799
Pemphigus erythematodes (Senear-Usher syndrome), 800
Subcutaneous fat necrosis of newborn (obstetric lipophagic granuloma), 609
Johnson, H. M.: Darier's disease, with possible nevoid changes on forehead, 448
Joints, Danlos' syndrome, 398
dermatolysis (Ehlers-Danlos syndrome), 1189
Juxta-Articular Nodules: See Nodes

Kahn, R. L.: Serologic verification test in diagnosis of latent syphilis, *817
Kahn Test: See also under Syphilis
temporarily positive Kahn and Wassermann reactions in infectious mononucleosis; report of case, 129
Kala-Azar: See Leishmaniasis
Kaposi's Sarcoma: See Sarcoma
Kaufman, S. R.: Generalized lupus erythematosus, vitamin deficiency (pseudopellagra), dermatomyositis or Addison's disease, 185
Seborrheic eczema, case for diagnosis, 982
Keil, H.: "Common mole"; its clinicopathologic relations and question of malignant degeneration, *214
Kelley, W. E.: Lupus erythematosus (extensive discoid), 183
Keloid: See also Cicatrix
extensive, following lightning, 392
localized bullous eruption with keloidal scarring (dermatitis artefacta?), 632
radiodermatitis; cellular keloid? 407
Kendrick, F. J.: Carcinoma of palate and alveolar process, 1168
Keratitis, clinical and experimental study of interstitial keratosis, 387
distinctive type of tuberculosis in Negro associated with tuberculous keratouveitis, 445
interstitial, 1122
interstitial; congenital syphilis; juvenile dementia paralytica, 167
rosacea with keratitis and corneal ulceration, 184
Keratoderma: See Keratosis
Keratosis: See also Ichthyosis
and arsenical pigmentation, 431
and arsenical pigmentation first manifested in patient at age of 6 years, 778
arsenical, case for diagnosis, 962
arsenical pigmentation and keratosis of palms and soles in patient with chronic myelogenous leukemia, 802
blennorrhagia, case for diagnosis, 607
follicularis; bullous disease; bullous Darier's disease (Pels and Goodman); familial benign pemphigus (Hailey and Hailey); herpetiform dermatitis repens (Ayers and Anderson), 1170
follicularis; congenital and familial hyperectodermosis, 933
follicularis (Darier's disease), 949, 1189
follicularis, Darier's disease in woman, 628
follicularis; Darier's disease, with possible nevoid changes on forehead, 448
keratoderma palmare et plantare hereditarium punctatum, 799
pathogenesis of hyperkeratoses in gold dermatitis, 578
pilaris, 177
Kerion: See Ringworm; Trichophytosis
Kerr, J. C.: Keratosis follicularis (Darier's disease), 949
Pigmentation from ferrous sulfate (copperas), 950
Sarcoid, case for diagnosis, 950
Kest, L. H.: Eczema of ear, associated with diphtheria-like organism; report of case, *1020
Kidneys, hypernephroma with cutaneous metastases, 576
Kierland, R. R.: Pyoderma faciale, *451
Kirby-Smith, H.: Vitamin D in treatment of acne, *835
Klauder, J. V.: Pityriasis rosea in infant, 450
Prevention of industrial dermatitis with reference to protective hand creams, soap and harmful role of some cleansing agents, *331
Kline Test: See under Syphilis
Klinge-Aschoff Nodule: See Rheumatic Fever
Klumpp, M. M.: Pityriasis rosea; review of literature and report of 219 cases, in 38 of which convalescent serum was used, *265
Urinary excretion of indoxyl (as indican) by infants with eczema, *113
Knowles, F. C.: Charles Nathaniel Davis, 572
Knuckle Pads: See under Fingers and Toes
Koilonychia: See Nails
Kolmer, J. A.: Influence of sodium dehydrocholate on therapeutic activity of neoarsphenamine; experiments with acute testicular syphilis of rabbits, *838
Kraurosis: See under Vulva
Kulchar, G. V.: Delayed positive reaction to patch test with nupercaine ointment, 1141
Heloderma simplex et annulare (Vörner), 992
Poikiloderma atrophicans vasculare, 991
Kunkel, F. E.: Sarcoid of Boeck, 1131

Labia Majora: See Vulva
LaFreniere, E. A.: Granuloma annulare, 424
Lain, E. S.: Lesions of oral cavity caused by physical and by physicochemical factors, *295
Lane, C. G.: Actinomycosis, 411
Lichen nitidus, 418
Lichen planus atrophicus or morphea, 424
Lupus erythematosus, 412
Mycosis fungoides, 410
Pemphigus, 412
Pseudo xanthoma elasticum? 424
Scar of oriental sore, 413
Spontaneous necrotic dermatitis (hemolytic streptococcus), 414
LaRocco, C. G.: Multiple epitheliomas; ulceration following use of "cancer paste," 1174
Larva Migrans: See Creeping Eruption
Larynx, gumma of, 979
lupus vulgaris of face; tuberculosis of buccal mucous membranes, pharynx and larynx; pulmonary tuberculosis, 1179
Laurence-Biedl Syndrome, lymphangioma and multiple hemangiomas in boy with adiposogenital dystrophy, 805
Lawless, T. K.: Scleroderma in chondrodystrophic person, 596
Lead, poisoning; effect of bismuth therapy on latent plumbism; preliminary report, *38
Legislation, protection of cosmetic consumer, *75
Legs, lymphangiectasia of vulva with edema of legs, 146
Lehndorff-Leiner Disease: See Erythema annulare rheumaticum
Leiner-Lehndorff Disease: See Erythema annulare rheumaticum
Leishmaniasis, oriental sore, 970
recidiva cutis; leishmanid, *874
scar of oriental sore, 413
Leprosy, case for diagnosis, 440
lepra with juxta-articular nodules, 789
Leukemia cutis, 941
lymphatic, 782
lymphatic; erythroderma psoriaticum cured with 30 per cent sulfur in petrolatum; coincident chronic lymphatic leukemia and recurrent herpes zoster, 136
lymphatic, with leukemids, 964
metastatic calcification; report of 2 cases, 385

Leukemia—Continued
myelogenous; arsenical pigmentation and keratosis of palms and soles in patient with chronic myelogenous leukemia, 802
Leukemid: See Leukemia, lymphatic
Leukocytes: See Leukemia
Leukoderma: See Vitiligo
Leukonychia: See under Nails
Leukoplakia: See Mouth; Vulva
Lever, W. F.: Role of vitamin C in various cutaneous diseases, *657
Levin, E. A.: Plastic induration of penis, case for diagnosis, 1148
Levin, O. L.: Erythroderma psoriaticum with tumor growths (epitheliomatous changes), 1180
Recklinghausen's disease; its elusive manifestations and internal relations, *480
Levine, B.: Capillaritis, 144
Congenital syphilis, 148
Hemiatrophy of face, 147
Macular atrophy, nonsyphilitic white spot disease or anetoderma erythematodes of Jadassohn, 147
Papilloma of palate probably due to red mercuric sulfide (cinnabar), 148
Scleroderma, 148
Syphilitic macular atrophy, 151
Urticaria pigmentosa, 146
Levy, H. A.: Nitritoid reaction to tryparsamide; report of case, *690
Lewis, G. M.: Cultural variations of Trichophyton purpureum (Bang) with discussion of recognizable features, *895
Delayed reaction to trichophytin, 945
Dermatophytosis, tinea barbae and tinea corporis due to Trichophyton purpureum, 938
Lichen planus of nail bed, 947
Parapsoriasis varioliformis, 945
Lichen nitidus, 418, 626, 1167
nitidus; hereditary ectodermal dysplasia of anhidrotic type associated with syndactylia and lichen nitidus, 946
planus, 787, 1128, 1146
planus atrophicus or morphea, 424
planus bullosus, 1138, 1139
planus, drug eruption or pityriasis lichenoides et varioliformis acuta, 765
planus following gold therapy, 763
planus, generalized, with lesions of palms and nails, 813
planus hypertrophicus, 620
planus hypertrophicus of legs with lupus erythematosus of face, 619
planus of eyelids, case for diagnosis, 1169
planus of nail bed, 947
planus or dermatitis medicamentosa from bismuth, 627
planus or lupus erythematosus, 411
planus, recurring, 793
planus sclerosus et atrophicus (Hallopeau), case for diagnosis, 178
planus sclerosus et atrophicus or dermatitis lichenoides chronica atrophicans (Csillag)? 422
planus; search for filtrable viruses in cutaneous diseases of unknown etiology, *261
planus starting on palms, 138
planus, ulcerated, of tongue, 933
planus with annular lesion on scalp, 796
planus, zosteriform, 620
sclerosus et atrophicus, 982
spinulosus, acquired, 1142
Lichenification: See Neurodermatitis
Light: See also Radiations; Ultraviolet Rays; etc.
passive transfer of light sensitivity; report of case, *889
significance of sex hormones in tanning of skin of women, 739
toxicity; eruptions and photosensitivity due to dyed fabrics, *1044
toxicity; photosensitizing agents; brief review of literature, 1112
Lightning, extensive keloid following lightning, 392
Lindsay, H. C. L.: Papulonecrotic tuberculid, case for diagnosis, 441
Lingua: See Tongue
Lipids: See Lipoids
Lipodystrophy: See Fat
Lipogranulomatosis and lupus erythematosus, 965
and subcutaneous adiponecrosis in infant, 576
Lipoidosis: See Schüller-Christian Syndrome; Xanthoma
Lipoids: See also Fat
imbibitio lipoidica collageni degenerati cutis, 180, 1193
necrobiosis lipoidica, 594
necrobiosis lipoidica diabeticorum, 151, 152, 594, 757, 959, 1143
Lips, cheilitis and dermatitis from tooth paste, *892
cheilitis due to deficiency of riboflavin, 1160
discrete inflammatory areas on vermilion border, 991
elephantiasis of lips and of male genitalia with reference to syphilis and lymphogranuloma venereum as etiologic factors, *11
lupus erythematosus involving lips; Addison's disease, 771
rate of ulceration of epitheliomas of skin and lip, *667
Lipstick, composition and dangers, *69
Livedo racemosa, case for diagnosis, 616
racemosa in syphilitic patient, 152
Liver Therapy: See Acne vulgaris; Pemphigus; etc.
Lukens, M.: Simplified complement fixation technic for diagnosis and treatment of syphilis; its sensitivity and specificity, *32
Lungs, Tuberculosis: See Tuberculosis, pulmonary
Lunsford, C. J.: Allergy due to cold, 1149
Pigmentation of skin of forehead, case for diagnosis, 1144
Lupus: See also Tuberculosis
cure in treatment of, 578
erythematosus, 412, 425, 436, 446, 584, 613, 618, 774, 947, 948, 980, 1117
erythematosus; acute arthritis associated with myocarditis simulating acute lupus erythematosus; report of case, *1053
erythematosus and lipogranulomatosis, 965
erythematosus bullosus or pemphigus, 797
erythematosus, bullous, 779
erythematosus, discoid, improved with germanin, 815
erythematosus, discoid, with dissemination, 419
erythematosus, disseminated, treated by sulfanilamide; report of 4 cases, *534
erythematosus disseminatus, 788
erythematosus disseminatus subacutus, 1130, 1161
erythematosus (extensive), 776
erythematosus, extensive discoid, 183
erythematosus, generalized; vitamin deficiency (pseudopellagra); dermatomyositis or Addison's disease, 185
erythematosus, group of cases of, 745
erythematosus involving lips; Addison's disease, 771
erythematosus; lichen planus hypertrophicus of legs with lupus erythematosus of face, 619
erythematosus of conjunctiva, 388
erythematosus or Besnier-Boeck-Schaumann disease, 416
erythematosus or lichen planus, 411
erythematosus or sarcoid, 760
erythematosus, subacute, 987
erythematosus, vitamin C in, *660
erythematosus with acrosclerosis, 1162
erythematosus with tuberculin reaction, 942
vulgaris, 174, 427, 744, 795

Lupus—Continued
vulgaris disseminatus, 392
vulgaris of face; tuberculosis of buccal mucous membranes, pharynx and larynx; pulmonary tuberculosis, 1179
vulgaris or syphilis? 426
vulgaris; roentgen ray dermatitis; tubular repair, 1138
vulgaris; temperature measurements in neighborhood of acrocyanosis and lupus vulgaris, 743
vulgaris (tumidus), 427
vulgaris; ulcers of tongue (unknown cause); osteomyelitis of right humerus, 624
vulgaris, vitamin C in, *660
Lymph Nodes: See also Lymphogranuloma
inguinal adenopathy of probable gonococcic origin, 932
lymphogranuloma venereum affecting simultaneously cervical and inguinal lymphatic glands; report of case, *557
pinta in Cuba; special clinical features of Cuban cases and discovery of spirochete in active lesions and in lymph glands, *463
Lymphadenopathy: See Lymph Nodes
Lymphangiectasia: See Lymphatic System
Lymphangioma and multiple hemangiomas in boy with adiposogenital dystrophy, 805
hemolymphangioma (preponderant hemangioma transformed to lymphangioma), 785
Lymphatic System: See also Lymph Nodes
lymphangiectasia of vulva with edema of legs, 146
Lymphedema: See Elephantiasis
Lymphoblastoma: See also Lymphogranuloma
Hodgkin's disease of skin or benign lymphadenoid granuloma (lymphocytoma), 623
or generalized neurodermatitis, 974
parakeratosis variegata, 603
Lymphocytes: See Leukemia, lymphatic
Lymphocytoma, lymphoblastoma (Hodgkin's disease of skin) or benign lymphadenoid granuloma (lymphocytoma), 623
two cases of miliary lymphocytoma or benign lymphadenoid granuloma of skin; lymphocytoma of face, 130
Lymphogranuloma: See also Granuloma
generalized erythroderma (Hodgkin's disease?), 753
Hodgkin's disease, 783
Hodgkin's disease, cutaneous and glandular, 756
Inguinale: See Granuloma inguinale; Lymphogranuloma Venereum
lymphoblastoma (Hodgkin's disease of skin) or benign lymphadenoid granuloma (lymphocytoma), 623
lymphogranulomatosis (Hodgkin's disease); treatment with sulfanilamide, 181
Lymphogranuloma Venereum, 977
affecting simultaneously cervical and inguinal lymphatic glands; report of case, *557
and syphilis (treated), 621
developmental form of virus, 935
elephantiasis of lips and of male genitalia with reference to syphilis and lymphogranuloma venereum as etiologic factors, *11
extragenital, on tongue and cervical nodes, 576
histologic study of Frei reaction papule, *120
morbilliform exanthem in, 936
skin test in lymphogranuloma inguinale [venereal lymphogranuloma], 386
treatment with sulfanilamide, 126
Lymphopathia Venereum: See Lymphogranuloma Venereum

McDonald, F.: Lymphangiectasia of vulva with edema of legs, 146
McDonnell, R. A.: Bullous lichen planus, 1139
Creeping eruption due to ancylostomiasis, 1139
McGovern, J. J.: Hemolytic jaundice with ulceration of skin, 498
MacKay, E. M.: Use of urea (carbamide) in treatment of warts (verruca vulgaris), *736
MacKee, G. M.: Roentgen dosage in dermatology expressed in international roentgens, *1
Madden, J. F.: Calcinosis cutis, 625
Depigmentation at site of chronic contact eczema, 621
Lichen planus hypertrophicus, 620
Lichen planus hypertrophicus of legs with lupus erythematosus of face, 619
Lupus vulgaris; ulcers of tongue (unknown cause); osteomyelitis of right humerus, 624
Lymphoblastoma (Hodgkin's disease of skin) or benign lymphadenoid granuloma (lymphocytoma), 623
Scrofuloderma; pulmonary tuberculosis, 624
Syphilis (treated); lymphogranuloma venereum? 621
Zosteriform lichen planus, 620
Xanthoma, 622
Magnesium hydroxide, fixed eruption from; polysensitivity, *707
Majocchi's Disease: See Purpura annularis telangiectodes
Malaria, Therapy: See under Dementia Paralytica; Syphilis
Malassezia Furfur: See Fungi
Mammary Gland: See Breast
Mapharsen: See Arsenic and Arsenic Compounds; Syphilis
Mapother, P.: Chromomycosis of face; report of case and study of causative organism, Phialophora verrucosa, *42
Marino, A. W. M.: Treatment of pruritus ani by tattooing with mercury sulfide, *521
Markham, F. S.: Inquiry into cause of pemphigus; is it virus disease? *78
Search for filtrable viruses in cutaneous diseases of unknown etiology, *261
Martin, W. B.: Erysipeloid; report of case, *1037
Maynard, M. T.-R.: Lichen spinulosus (acquired), 1142
Pseudopelade, 985
Vitamin therapy in dermatology with reference to vitamin D in treatment of acne and of diseases due to altered usage of calcium, *842
Mecholyl: See Choline and Choline Derivatives
Medulla Oblongata, trophic ulcers of face and neck due to syringobulbia, 153
Meige's Disease: See Edema
Melanin: See Addison's Disease; Pigmentation
Melanoblastoma, 1167
cutaneous (melanosarcoma), 141
Melanoma, blue nevus (Jadassohn-Tieche); its distinction from ordinary moles and malignant melanomas, 1111
malignant, 433
of urethra, 1110
Melanosarcoma, cutaneous melanoblastoma, 141
Melanosis: See Pigmentation
Memmesheimer, A.: Sudoriparous glands; sweat, *1086
Mendelsohn, H. V.: Fixed eruption with extracutaneous manifestation due to mapharsen; report of case, *509
Meningomyelitis syphilitica with muscular atrophy, 158
Menstruation, desensitization in treatment of menstrual intoxication and other allergic symptoms, 130
Mercury, action of sulfur-containing compounds in arsenical and mercurial poisoning with review of use of sodium thiosulfate in dermatologic practice and report of experiments on rabbits with sodium thiosulfate, sodium p-sulfhydril phenyl sulfonate and sodium formaldehyde sulfoxylate, *308
papilloma of palate probably due to red mercuric sulfide (cinnabar), 148

Metals: See Lead; Mercury; Silver; etc.
Metasyphilis: See Syphilis, congenital
Methylrosaniline, sensitivity to gentian violet (methylrosaniline), *122
Therapy: See Ringworm
Miller, H. E.: Arsenical pigmentation and keratoses, 431
Coccidioidal granuloma, 990
Pemphigus, 985
Pemphigus of conjunctiva, 429
Miller, J. L.: Tertiary syphilis; aneurysm of ascending portion of aorta, 161
Milroy's Disease: See Edema
Mint, occupational dermatitis due to; report of 2 cases, *503
Mitchell, D. S.: Atopic eczema with bilateral cataract, 402
Eczema seborrhoicum, 404
Kaposi's sarcoma, 404
Lupus vulgaris disseminatus, 392
Phenolphthalein pigmentation, case for diagnosis, 399
Syphilis with negative serologic changes; exfoliative dermatitis, 391
Xanthomatosis (Schüller-Christian disease), 400
Mitchell, J. H.: Basal cell epithelioma of glans penis, case for diagnosis, 781
Mycosis fungoides, 581, 582
Psoriasis pustulosa, case for diagnosis, 1157
Mites, dermatitis, *831
Molds: See Fungi
Moles: See Nevi
Monilia albicans, onychomycosis due to, 951
Moniliasis, case for diagnosis, 176
or pemphigus vulgaris, 1136
or tertiary syphilis, 767
Mononucleosis, infectious, temporarily positive Kahn and Wassermann reactions in; report of case, 129
Montgomery, D. W.: Fistulas from dead teeth simulating dermal epithelioma, *378
Montgomery, H.: Acrodermatitis chronica atrophicans, 750
Angioma serpiginosum, case for diagnosis, 751
Dermatitis herpetiformis, 755
Generalized erythroderma (Hodgkin's disease?), 753
Group of cases of lupus erythematosus, 745
Group of cases of pyoderma with various systemic manifestations, 752
Hodgkin's disease, cutaneous and glandular, 756
Localized and linear scleroderma, atrophic and indurated, 749
Lupus vulgaris, 744
Perforating gumma of soft palate and nasal septum; asymptomatic neurosyphilis, 751
Rosacea-like tuberculid of Lewandowsky, 744
Sarcoid or lupus erythematosus, 760
Seborrheic eczema, 754
Senile pruritus (neurodermatitis?); hemangioendothelioma of left foot (Kaposi's sarcoma?), 754
Tinea capitis; kerion celsi, 748
Tinea capitis; kerion celsi; trichophytid, 748
Moore, M.: Chromomycosis of face; report of case and study of causative organism, Phialophora verrucosa, *42
Malassezia furfur, cause of tinea versicolor; cultivation of organism and experimental production of disease, *253
Morphea Guttata: See Scleroderma
Morrissey, M. J.: Acne varioliformis, 1135
Dermatitis herpetiformis, 1136
Mouth: See also Stomatitis; etc.
hypertrophic nevus of scalp, face and oral mucosa, 1191
lesions of oral cavity caused by physical and by physicochemical factors, *295
leukoplakia and allied mouth conditions, 1111
lupus vulgaris of face; tuberculosis of buccal mucous membranes, pharynx and larynx; pulmonary tuberculosis, 1179
Mouth—Continued
pemphigus of long duration in mouth, with acute exacerbation on skin; improvement following large doses of vitamin D, 630
perioral pigmentation due to bismuth? 966
recurrent erythema multiforme of oral mucosa associated with progressive Fordyce's disease, 1192
recurrent ulcerations for 17 years, case for diagnosis, 1183
thickening and exfoliation of mucous membrane, case for diagnosis, 439
tuberculosis, 738
Mucous Membrane: See Mouth; Nose; etc.
Muir, K. B.: Action of sulfur-containing compounds in arsenical and mercurial poisoning with review of use of sodium thiosulfate in dermatologic practice and report of experiments on rabbits with sodium thiosulfate, sodium p-sulfhydril phenyl sulfonate and sodium formaldehyde sulfoxylate, *308
Muller, J.: Atrophia cutis maculosa, case for diagnosis, 415
Murray, M.: Chromidrosis; report of case, *379
Mutscheller, A.: Absorption of roentgen rays by skin; experimental determinations, *87
Mycosis: See also Actinomycosis; Blastomycosis; Moniliasis; Ringworm; Stomatitis; Tinea; Trichophytosis; etc.
cutaneous; dermatophytosis, tinea barbae and tinea corporis due to Trichophyton purpureum, 938
cutaneous; generalized dermatophytid after intracutaneous tests with trichophytin in dilutions of 1:30 and 2:0; combination of erysipelas and epidermophytosis, 983
cutaneous; poikiloderma atrophicans vasculare, dermatomycosis and onychomycosis, 581
cutaneous; pruritus ani and its relationship to seborrheic eczema and dermatophytosis, *699
cutaneous; psoriasis, bacterid or dermatophytosis with dermatophytide? 1140
fungoides, 145, 155, 403, 410, 581, 582, 583, 1126, 1182
fungoides and generalized exfoliative psoriasis, *119
fungoides atrophicans following premycotic stage which resembles pruritus with lichenification, 577
fungoides (exfoliative dermatitis; tumors of face), 970
fungoides with pernicious anemia, 616
Myelitis, acute syphilitic transverse myelitis; clinical study and report of case, *1078
tertiary syphilis; transverse myelitis following administration of bismuth compound, 160
Myelosis, Aleukemic: See Leukemia, myelogenous
Myocarditis, acute arthritis associated with myocarditis simulating acute lupus erythematosus; report of case, *1053
Myositis ossificans, 410
Myxedema, nodular, in case of thyrotoxicosis, 422

Nails, congenital and familial hyperectodermosis, 933
diseases; report of cases of onycholysis, *98
generalized lichen planus with lesions of palms and nails, 813
Kaposi's sarcoma and leukonychia, 618
koilonychia, 784
lichen planus of nail bed, 947
nail polishes; composition and dangers, *70
onychia due to handling sugar, 142
onychomycosis due to Monilia albicans, 951
pachyonychia congenita; dermatitis of hands, 772
poikiloderma atrophicans vasculare, dermatomycosis and onychomycosis, 581
vitamin D in treatment of leukonychia, *846
Nares: See Nose

Necrobiosis lipoidica, 594
lipoidica diabeticorum, 151, 152, 594, 757, 959, 1143
Neoarsphenamine: See Arsphenamines; and under names of various diseases
Neoplasms: See Cancer; Sarcoma; Tumors
Nerves: See also Neuritis; Paralysis
optic, beginning atrophy of, 1123
optic, bilateral atrophy of, 1122
optic; Erb's spastic paraplegia; primary atrophy of optic nerve, 163
optic, primary atrophy of, 1122
optic, primary atrophy of; juvenile tabes dorsalis, 447
optic, syphilitic atrophy of, 971
tabes (Charcot knee; atrophy of optic nerve; chorioretinitis), 171
Nervous System: See also Brain; Nerves; etc.
pathology of unilateral hyperhidrosis of face of central origin, 742
Recklinghausen's disease; its elusive manifestations and internal relations, *480
sympathetic, role in reactions to arsenical chemotherapy, 740
Syphilis: See Syphilis
Netherton, E. W.: Cutaneous hypersensitivity to triethanolamine; report of 2 cases, *729
Elephantiasis of lips and of male genitalia with reference to syphilis and lymphogranuloma venereum as etiologic factors, *11
Necrobiosis lipoidica diabeticorum, 151
Pityriasis lichenoides chronica, 150
Neuhauser, I.: Sarcoid of Boeck, 1172
Neuritis, retrobulbar, with pellagra in Nigeria, 740
Neurodermatitis, 395
atopic dermatitis, 756, 1149
atopic dermatitis or bacterial dermatitis, 417
generalized, or lymphoblastoma, 974
gigantea, case for diagnosis, 779
mycosis fungoides atrophicans following premycotic stage which resembles pruritus with lichenification, 577
senile pruritus (neurodermatitis?); hemangioendothelioma of left foot (Kaposi's sarcoma?), 754
vitamin D in treatment of psoriasis and atopic eczema, *846
Neurofibromatosis, von Recklinghausen's disease, 178, 593
Recklinghausen's disease; its elusive manifestations and internal relations, *480
unusual form of morbus Recklinghausen with painful, occasionally ulcerating hard subcutaneous neurofibromas and severe ocular changes but no other signs of Recklinghausen's disease, 936
Neuroses and Psychoneuroses, neurotic excoriations; case for diagnosis, 966
Neurosyphilis: See Syphilis
Nevi: See also Angioma
blue (Jadassohn), 959
blue (Jadassohn-Tieche); its distinction from ordinary moles and malignant melanomas, 1111
"common mole"; its clinicopathologic relations and question of malignant degeneration, *214
Darier's disease, with possible nevoid changes on forehead, 448
hypertrophic, of scalp, face and oral mucosa, 1191
minor congenital ectodermal cutaneous defect in naevus linearis distribution, 132
hereditary familial telangiectasia, 938
Newborn Infants: See Infants, newborn
Nickel, dermatitis due to safety pins; interesting sequence of sensitization due to contact with nickel, *568
dermatitis eczematosa (contact type) due to, 815
dermatitis from spectacle frames, 446
Nicolas-Favre Disease: See Lymphogranuloma Venereum
Niles, H. D.: Atrophic glossitis, case for diagnosis, 769
Leukoderma acquisitum centrifugum (Sutton), 1190
Pityriasis rosea; review of literature and report of 219 cases, in 38 of which convalescent serum was used, *265
Nipples: See Breast
Nitritoid Crises: See Tryparsamide
Nodes: See also Erythema nodosum; Panniculitis; Rheumatic Fever; etc.
lepra with juxta-articular nodules, 789
Noma: See Stomatitis, gangrenous
Nose, perforating gumma of soft palate and nasal septum; asymptomatic neurosyphilis, 751
tuberculous granuloma; case for diagnosis, 435
Nostrums; multiple epitheliomas; ulceration following use of "cancer paste," 1174
Nupercaine, delayed positive reaction to patch test with nupercaine ointment, 1141

Obermayer, M. E.: Bullous disease; bullous Darier's disease (Pels and Goodman); familial benign pemphigus (Hailey and Hailey); herpetiform dermatitis repens (Ayers and Anderson), 1170
Cutaneous metastatic carcinoma originating from osteomyelitic cavities, *545
Histiocytoma, case for diagnosis, 1151
Lichen planus of eyelids, case for diagnosis, 1169
Parakeratosis variegata (lymphoblastoma?), 603
Pityriasis rubra pilaris, 599
Poikiloderma atrophicans vasculare, dermatomycosis and onychomycosis, 581
Prurigo of Hebra or pigmentation? 600
Tuberculosis verrucosa cutis, 779

OBITUARIES:
Davenport, Kenneth Mercereau, 1200
Davis, Charles Nathaniel, 572
Fisher, George M., 634
Kirby-Smith, Joseph Lee, 123
Simpson, Charles Augustus, 188, 382
Taussig, Laurence R., 634, 930

Occupational Diseases: See Industrial Diseases
Old Age, senile elastosis and acrodermatitis chronica atrophicans, 168
O'Leary, P. A.: Acrodermatitis chronica atrophicans, 750
Angioma serpiginosum, case for diagnosis, 751
Dermatitis herpetiformis, 755
Dermatomyositis; study of 40 cases, *1001
Generalized erythroderma (Hodgkin's disease?), 753
Group of cases of lupus erythematosus, 745
Group of cases of pyoderma with various systemic manifestations, 752
Hodgkin's disease, cutaneous and glandular, 756
Localized and linear scleroderma, atrophic and indurated, 749
Lupus vulgaris, 744
Perforating gumma of soft palate and nasal septum; asymptomatic neurosyphilis, 751
Pyoderma faciale, *451
Rosacea-like tuberculid of Lewandowsky, 744
Sarcoid or lupus erythematosus, 760
Seborrheic eczema, 754
Senile pruritus (neurodermatitis?); hemangioendothelioma of left foot (Kaposi's sarcoma?), 754
Tinea capitis; kerion celsi, 748
Tinea capitis; kerion celsi; trichophytid, 748
Oliver, E. A.: Arsenical dermatitis (chronic), 590
Leukoderma (occupational), 587
Omens, D. V.: Acarophobia and tumors due to camphor in oil, 601
Atypical psoriasis, case for diagnosis, 777
Boeck's sarcoid, 592

Omens, D. V.—Continued
Bullous lupus erythematosus, 779
Condom dermatitis, 1160
Fixed eruption, case for diagnosis, 1155
Lichen planus, 787
Lupus erythematosus with acrosclerosis, 1162
Recklinghausen's disease, 593
Sarcoid of Boeck, 1154
O'Neill, C. W.: Contact dermatitis from rubber and alcohol, 1125
Granuloma inguinale, 1127
Juvenile disseminated xanthomatosis, 1134
Leukoderma, 1132
Lichen planus bullosus, 1138
Lupus vulgaris; roentgen ray dermatitis; tubular repair, 1138
Psoriasis; bacterid? dermatophytosis? with dermatophytide? 1140
Onychia: See Nails
Onycholysis: See under Nails
Onychomycosis: See Nails
Oppenheim, M.: Lichen nitidus, 1167
Oriental Sore: See Leishmaniasis
Ormsby, O. S.: Case for diagnosis, 589
Juvenile xanthoma, 586
Sarcoid (Schaumann's syndrome), 591
Orthodichlorobenzene: See Chlorobenzene
Osler's Disease: See Telangiectasis
Osteitis, syphilitic; late secondary syphiloderm, 173
Tuberculous: See Bones, tuberculosis
Osteomatosis cutis, *562
Osteomyelitis, cutaneous metastatic carcinoma originating from osteomyelitic cavities, *545
lupus vulgaris; ulcers of tongue (unknown cause); osteomyelitis of right humerus, 624
Osteoperiostitis of femur, 158
Ostwald, E.: Acne conglobata, 1150
Oulmann, L.: Lichen planus starting on palms, 138
Ovary, clinical evaluation of ovarian responses to gonadotropic therapy, 573
tumors; case of arrhenoblastoma which simulated Cushing's disease, 385

Pace, E. R.: Lupus erythematosus (extensive), 776
Paget's Disease of Breast: See under Breast
Pain, painful subcutaneous tubercle (tuberculum dolorosum), 1110
Palate, carcinoma of palate and alveolar process, 1168
papilloma probably due to red mercuric sulfide (cinnabar), 148
perforating gumma of soft palate and nasal septum; asymptomatic neurosyphilis, 751
tuberculoma of palate and right angle of mouth; miliary pulmonary tuberculosis, 1179
Panniculitis, relapsing febrile nonsuppurative; report of 2 cases, *681
Papaverine: See Raynaud's Disease
Papilloma of palate probably due to red mercuric sulfide (cinnabar), 148
papillomatosis cutis, 935
Parakeratosis Variegata: See Parapsoriasis
Paralysis, facial, due to early syphilis of central nervous system; granuloma inguinale, 154
spinal; Erb's spastic paraplegia; primary atrophy of optic nerve, 163
Paraplegia: See Paralysis, spinal
Parapsoriasis, 610
en plaque type? 441
guttata, 976
lichenoides chronica, 1197
parakeratosis variegata (lymphoblastoma?), 603
varioliformis, 945
varioliformis acuta, 967, 976
Pariser, H. P.: Rosacea with keratitis and corneal ulceration, 184
Parkes-Weber Syndrome: See Varicose Veins
Patch Test: See Anaphylaxis and Allergy
Peck, S. M.: Discoid lupus erythematosus improved with germanin, 815
Recurrent bullous eruption (Duhring's disease?) treated with trichophytin, 812
Recurrent ulcerations of mouth for 17 years, case for diagnosis, 1183
Pellagra: See also Vitamins, B
case in British Columbia, 1112
dermatitis with depigmentation, 1152
of Kentucky mountain folk; ambulatory treatment with nicotinic acid, 1113
vitiligo with superimposed pellagra, 625
with retrobulbar neuritis in Nigeria, 740
Pemphigus, 412, 985
bullous disease; bullous Darier's disease (Pels and Goodman); familial benign pemphigus (Hailey and Hailey); herpetiform dermatitis repens (Ayers and Anderson), 1170
erythema multiforme or dermatitis herpetiformis, 444
erythematodes, case for diagnosis, 791
erythematodes (Senear-Usher syndrome), 800, 808
experimental studies on 34 patients, *359
foliaceus (fogo salvagem); endemic disease of state of São Paulo (Brazil), *858
inquiry into cause; is it virus disease? *78
of conjunctiva, 429
of long duration in mouth, with acute exacerbation on skin; improvement following large doses of vitamin D, 630
of Senear-Usher or dermatitis medicamentosa, 795
or epidermolysis bullosa, 434
or lupus erythematosus bullosus, 797
Senear-Usher syndrome, 405
treatment with liver preparations, 742
vitamin D in treatment of, *845
vulgaris, 984
vulgaris; cases of familial and of conjugal pemphigus vulgaris; use of transfusions of blood from cured patients, *1073
vulgaris or moniliasis, 1136
Penis, atrophic process of glans penis with epithelioma, 432
balanitis; case for diagnosis, 181
balanitis xerotica obliterans, 629
basal cell epithelioma of glans penis? 781
Bowen's disease of, 442
elephantiasis of lips and of male genitalia with reference to syphilis and lymphogranuloma venereum as etiologic factors, *11
epithelioma of, 629
erythroplasia of Queyrat, 1192
plastic induration of, 1148
unusual location of glomus tumor; report of 2 cases, 130
Perfume: See also Cologne Water
composition and dangers, *73
Periarteritis, ainhum or syphilis, 443
nodosa, 384
Periostitis, recent periostitis developing during treatment of late syphilis; iridocyclitis (old) with secondary glaucoma, 157
Periphlebitis nodularis necrotisans; attempt at definition and classification, *55
Persky, A. M.: Recurrent cellulitis resulting in chronic lymphedema, 171
Perspiration: See under Sweat; Sweat Glands
Petges-Clijat Disease: See Poikiloderma
Phagedena, geometric (Brocq), 1195
Phalanges: See Fingers and Toes
Pharynx, lupus vulgaris of face; tuberculosis of buccal mucous membranes, pharynx and larynx; pulmonary tuberculosis, 1179
Phenolphthalein pigmentation (from ex-lax), case for diagnosis, 399
Phialophora Verrucosa: See Fungi
Phillips, H. T.: Keratosis follicularis (Darier's disease), 949
Pigmentation from ferrous sulfate (copperas), 950
Sarcoid, case for diagnosis, 950

Photodermatosis: See Light, toxicity
Photography, natural color photography of skin; new apparatus for proper illumination, *527
Photosensitization: See Light; Ultraviolet Rays
Physostigmine, contact dermatitis due to physostigmine salicylate, 147
Pigmentation: See also Addison's Disease; Hemochromatosis; Nevi; Schamberg's Disease; etc.
arsenical dermatitis with pigmentation and alopecia, 761
fixed, 603
from ferrous sulfate (copperas), 950
of skin of forehead, case for diagnosis, 1144
or prurigo of Hebra, 600
perioral, due to bismuth, 966
photosensitizing agents; brief review of literature, 1112
significance of sex hormones in tanning of skin of women, 739
universal depigmentation of skin, with areas of hyperpigmentation, 444
Pillsbury, D. M.: Localized bullous eruption with keloidal scarring (dermatitis artefacta?), 632
Pustular psoriasis or "id"? 186
Pines, C.: Syphilitic atrophy of optic nerve, 971
Pinta in Cuba; special clinical features of Cuban cases and discovery of spirochete in active lesions and in lymph glands, *463
White: See Vitiligo
Pituitary Body, tumors; case of arrhenoblastoma which simulated Cushing's disease, 385
Pityriasis lichenoides chronica, 150
lichenoides et varioliformis acuta, drug eruption or lichen planus, 765
rosea in infant, 450
rosea; review of literature and report of 219 cases, in 38 of which convalescent serum was used, *265
rosea; search for filtrable viruses in cutaneous diseases of unknown etiology, *261
rubra pilaris, 599
Versicolor: See Tinea versicolor
Pityrosporum: See Fungi
Plumbism: See Lead, poisoning
Poikiloderma atrophicans vasculare, 991
atrophicans vasculare, dermatomycosis and onychomycosis, 581
of Civatte, 809
poikilodermatomyositis, case for diagnosis, 790
poikilodermatomyositis (Petges-Clijat), 1187
reticulated pigmented poikiloderma of face and neck (Civatte), 631
Poison Ivy: See Rhus, poisoning
Poisons and Poisoning: See Dyes; and under names of substances, as Lead; Mercury; etc.
Poliosis: See Hair, color of
Polishes, nail polishes; composition and dangers, *70
Pollen: See also Asthma; Ragweed
sensitization of skin induced by turbinal pollen application, resulting in facial acne, 1114
Powders, toilet powders; composition and dangers, *66
Pratt, A. G.: Kaposi's sarcoma (?) in American boy aged 15, 981
Pregnancy, complications due to arsenical therapy in syphilitic pregnant women; report of 7 maternal deaths, 128
laws requiring premarital and pregnancy tests for syphilis; reasons for and against, 1112
metastatic calcification; report of 2 cases, 385
Prepuce: See Penis
Pringle-Bourneville's Disease: See Sclerosis, tuberous
Prostate, cancer; metastases of scalp simulating turban tumors, *639
Protein, cutaneous absorption; direct technic for demonstrating percutaneous absorption of antigens, *692
Prurigo of Hebra, 406
of Hebra or pigmentation, 600
Pruritus ani and its relationship to seborrheic eczema and dermatophytosis, *699
ani, treatment by tattooing with mercury sulfide, *521
mycosis fungoides atrophicans following premycotic stage which resembles pruritus with lichenification, 577
senile (neurodermatitis); hemangioendothelioma of left foot (Kaposi's sarcoma?), 754
Pseudoatrophoderma Colli (?): See Atrophy
Pseudopelade: See Alopecia cicatrisata
Pseudopellagra, generalized lupus erythematosus, vitamin deficiency (pseudopellagra), dermatomyositis or Addison's disease, 185
Pseudoxanthoma elasticum? 424
Psoriasis and atopic eczema, vitamin D in treatment of, *846
and granuloma annulare, 133
atypical, case for diagnosis, 777
bacterid or dermatophytosis with dermatophytide? 1140
case for diagnosis, 784
erythroderma psoriaticum with tumor growths (epitheliomatous changes), 1180
generalized exfoliative psoriasis and mycosis fungoides, *119
latent, case activated by roentgen irradiation, 935
papular, case for diagnosis, 949
pustular, or bacterid? 580
pustular, or "id"? 186
pustulosa, case for diagnosis, 1157
search for filtrable viruses in cutaneous diseases of unknown etiology, *261
treatment with concentrated viosterol under auspices of Cincinnati Society of Dermatology and Syphilology, *664
vitamin C in, *659
Ptosis: See under Eyelids
Public health problems in control of syphilis, *904
Purpura annularis telangiectodes, 138, 437, 982
due to ingestion of cinchophen, 1176
haemorrhagica; caution in arsphenamine therapy (report of case of purpura haemorrhagica following treatment with neoarsphenamine), 1111
haemorrhagica following use of sedormid; 2 attacks in 1 person, 129
simplex, 612
vitamin C in, *661
Pyoderma and chronic ulcerative colitis, 752
and hidrosadenitis with colitis, 753
eruptions and photosensitivity due to dyed fabrics, *1044
faciale, *451
gangraenosum, 1156
gangraenosum of neck and thighs without colitis, 752
gangraenosum treated with sulfanilamide; report of case, *711
group of cases with various systemic manifestations, 752

Quackery: See Nostrums
Quarrier, S. S.: Reticulum cell sarcoma; report of case with pronounced cutaneous manifestations, *722
Quinine, Therapy: See Stomatitis, gangrenous

Radiations: See also Roentgen Rays; Ultraviolet Rays; etc.
histamine-like effect of irradiated or inflammatory skin, 741
Radiodermatitis: See under Roentgen Rays
Radium, Therapy: See under names of various diseases

Ragweed, treatment of ragweed dermatitis; report of 7 cases of successful treatment with absolute alcohol extract of ragweed leaves, *551
Rattner, H.: Erythema multiforme or drug eruption, 786
Hodgkin's disease, 783
Oil tumors, case for diagnosis, 1153
Reticuloendotheliosis, case for diagnosis, 780
Sarcoid of Boeck, 1154, 1172
Raynaud's Disease, treatment with papaverine intravenously, 383
von Recklinghausen's Disease: See Neurofibromatosis
Rectum, granuloma inguinale of vaginal wall and rectum with profuse hemorrhage, 801
Reinhard, M. C.: Qualitative measurements of low voltage shock-proof x-ray tubes, *718
Rendu-Osler's Disease: See Telangiectasis
Reticuloendothelial System: See also Jaundice; Mononucleosis; etc.
diseases; reticuloendotheliosis, 780, 937
reticulum cell sarcoma; report of case with pronounced cutaneous manifestations, *722
Reticuloendotheliosis: See Reticuloendothelial System, diseases
Retina, pseudoxanthoma elasticum? 424
Retinochoroiditis, chorioretinitis, 1122
tabes (Charcot knee; atrophy of optic nerve; chorioretinitis), 171
Reynolds, H. S.: Acne varioliformis, 1135
Dermatitis herpetiformis, 1136
Rheumatic Fever, erythema annulare rheumaticum (Lehndorff-Leiner); report of case, *515
rheumatic subcutaneous nodule and simulating lesions, 574
rheumatic symptoms in patients with erythema nodosum, 937
Rhus, poisoning; pigmentation from ferrous sulfate (copperas), 950
poisoning; sensitivity to poison ivy, 739
poisoning; studies on sensitization of animals with simple chemical compounds, 573
Riboflavin: See Vitamins, B
Ricchiuti, J. F., Jr.: Treatment of ragweed dermatitis; report of 7 cases of successful treatment with absolute alcohol extract of ragweed leaves, *551
Ringworm: See also Mycosis, cutaneous; Tinea; Trichophytosis; etc.
tinea capitis; kerion celsi, 748
treatment of ringworm of scalp with gentian violet, *370
Ritter's Disease: See Dermatitis, exfoliative
Robinson, S. S.: Lupus erythematosus involving lips; Addison's disease, 771
Roentgen Rays: See also Radiations
absorption by skin; experimental determinations, *87
case of latent psoriasis activated by roentgen irradiation, 935
lupus vulgaris; roentgen ray dermatitis; tubular repair, 1138
qualitative measurements of low voltage shock-proof x-ray tubes, *718
radiodermatitis; cellular keloid? 407
radiodermatitis of hands with malignant degeneration, 149
Therapy: See under names of organs, regions and diseases
vitamin D in scleroderma and roentgen ray sclerosis, *844
Roentgenotherapy, roentgen dosage in dermatology expressed in international roentgens, *1
Rogers, J. D.: Dermatitis herpetiformis perstans, 773
Multiple benign epithelioma tosis, 774
Rokstad, I.: New modification of patch test (chamber method), *649
Ronchese, F.: Lupus vulgaris or syphilis, 426
Lupus vulgaris (tumidus), 427
Metastases of scalp simulating turban tumors, *639
Rosen, I.: Darier's disease, 1189
Livedo racemosa, case for diagnosis, 616
Mycosis fungoides, 1182
Rosenbach's Disease: See Erysipeloid
Rosenberg, J.: Erythema exsudativum multiforme (Hebra) with conjunctivitis and stomatitis, *1066
Rosenberg, L.: Erythema exsudativum multiforme (Hebra) with conjunctivitis and stomatitis, *1066
Rosenfeld, H.: Recurrent bullous eruption (Duhring's disease) treated with trichophytin, 812
Rostenberg, A.: Acne urticata polycythaemica, 807
Tertiary syphilis and syphilitic macular atrophy, 811
Roussy-Darier Sarcoid: See Sarcoid
Rubber, condom dermatitis, 1160
contact dermatitis from rubber and alcohol, 1125
Rule, A. M.: Influence of sodium dehydrocholate on therapeutic activity of neoarsphenamine; experiments with acute testicular syphilis of rabbits, *838
Russo, J. J.: Fixed eruption from magnesium hydroxide; polysensitivity, *707

Sáenz, B.: Pinta in Cuba; special clinical features of Cuban cases and discovery of spirochete in active lesions and in lymph glands, *463
Safety pins, dermatitis due to; interesting sequence of sensitization due to contact with nickel, *568
Sams, W. M.: Occupational dermatitis due to mint; report of 2 cases, *503
Sarcoid, Besnier-Boeck-Schaumann disease, 759
Boeck's, 573, 592, 1131, 1154, 1172
case for diagnosis, 950
hypodermic sarcoid, 401
lupus erythematosus or Besnier-Boeck-Schaumann disease, 416
of Darier and Roussy, 759
or lupus erythematosus, 760
osteitis tuberculosa multiple cystoides and sarcoid lesions, 574
sarcoidosis, 793
Schaumann's syndrome, 591
Spiegler-Fendt, 988
Sarcoma: See also Cancer; Tumors; and under names of tumors, as Melanosarcoma; etc.
Kaposi's, 404
Kaposi's, and leukonychia, 618
Kaposi's (?) in American boy aged 15, 981
Kaposi's, with lesions of face, 596
reticulum cell sarcoma; report of case with pronounced cutaneous manifestations, *722
senile pruritus (neurodermatitis?); hemangioendothelioma of left foot (Kaposi's sarcoma?), 754
Sayer, A.: Generalized lichen planus with lesions of palms and nails, 813
Glossitis rhombica mediana; interstitial atrophic syphilitic glossitis, 979
Lymphoblastoma or generalized neurodermatitis, 974
Scabies, acarophobia and tumors due to camphor in oil, 601
Scalp, Diseases: See under names of diseases, as Mycosis; Ringworm; Seborrhea; Tinea; etc.
erythrokeratoderma of scalp and familial "trichorrhexis nodosa," 934
hypertrophic nevus of scalp, face and oral mucosa, 1191
lichen planus with annular lesion on scalp, 796
metastases of scalp simulating turban tumors, *639
treatment of ringworm of scalp with gentian violet, *370
wood tick simulating pedunculated tumor, *887

Scars: See Cicatrix
Schamberg's Disease, case for diagnosis, 789
progressive pigmentary dermatosis, 952
unilateral, 170
Schaumann's Sarcoid: See Sarcoid
Scheer, M.: Epidermolysis bullosa hereditaria, 137
Hereditary familial telangiectasia, case for diagnosis, 1189
Nevus or hereditary familial telangiectasia, 938
Urticaria from heat, 1191
Schiller, A. E.: Beginning atrophy of optic nerves, 1123
Bilateral atrophy of optic nerves, 1122
Chorioretinitis, 1122
Eczema seborrhoicum, with unusual persistent localization, 1121
Generalized scleroderma, 1119
Interstitial keratitis, 1122
Lupus erythematosus, 1117
Natural color photography of skin; new apparatus for proper illumination, *527
Primary atrophy of optic nerve, 1122
Scleroderma, 1118, 1119
Sycosis vulgaris, 1116
Schistosomiasis, methods for controlling schistosome dermatitis (use of copper carbonate to kill snails), 1113
Schmidt, O. E. L.: Arsenical pigmentation and keratosis of palms and soles in patient with chronic myelogenous leukemia, 802
Capillaritis, case for diagnosis, 803
Infectious eczematoid dermatitis treated with sulfanilamide, 803
Schmitt, C. L.: Lupus erythematosus, 948
Pseudopelade (Brocq), 948
Schoch, A. G.: Erysipeloid of Rosenbach successfully treated with sulfanilamide, *570
New acne stilet, *1108
Schoff, C. E.: Pseudoatrophoderma colli (?), 430
Schonberg, I. L.: Congenital syphilis, 148
Generalized exfoliative psoriasis and mycosis fungoides, *119
Mycosis fungoides, 145
Schorr, H. C.: Sarcoid (Schaumann's syndrome), 591
Schüller-Christian Syndrome, juvenile xanthomatosis (possible Hand-Schüller-Christian disease), 585
xanthomatosis, 400
Schwartz, G.: Lichen planus sclerosus et atrophicus or dermatitis lichenoides chronica atrophicans (Csillag)? 422
Schwartz, L.: Leukoderma (occupational), 587
Schwartz, W. F.: Atrophic arthritis with subcutaneous nodules and pulmonary tuberculosis, 144
Atrophic arthritis with subcutaneous nodules and tertiary syphilis, 143
Facial paralysis due to early syphilis of central nervous system; granuloma inguinale, 154
Juvenile tabes dorsalis, 154
Mycosis fungoides, 155
Trophic ulcers of face and neck due to syringobulbia, 153
Xanthoma diabeticorum chiefly limited to palms, 139
Schweig, J.: Generalized Trichophyton purpureum infection simulating dermatitis herpetiformis; report of case, *732
Scleredema: See Scleroderma
Scleroderma, 148, 1118, 1119
and roentgen ray sclerosis, vitamin D for, *844
cutaneous and subcutaneous calcinosis; scleroderma (right cheek)? 1140
esophageal lesions in, 934
generalized, 1119
in chondrodystrophic person, 596
lichen planus atrophicus or morphea, 424
localized and linear scleroderma, atrophic and indurated, 749
Scleroderma—Continued
localized atrophy of skin and subcutaneous tissue (hemiatrophia faciei? scleroderma?), 437
lupus erythematosus with acrosclerosis, 1162
macular atrophy, nonsyphilitic white spot disease or anetoderma erythematodes of Jadassohn, 147
metabolic cutaneous calcinosis; report of case with scleroderma, *864
or Bazin's disease, 951
progressive type, 419
Sclerosis, tuberous; Bourneville-Pringle syndrome, 791
Scrofula: See Tuberculosis
Scrofuloderma: See Tuberculids; Tuberculosis
Scrotum, elephantiasis of lips and of male genitalia with reference to syphilis and lymphogranuloma venereum as etiologic factors, *11
Seasons, winter eczema, *569
Sebaceous Glands, basal cell epithelioma located over sebaceous cyst, 978
Seborrhea, eczema seborrhoicum, 404
eczema seborrhoicum, with unusual persistent localization, 1121
pruritus ani and its relationship to seborrheic eczema and dermatophytosis, *699
seborrheic eczema, 754, 982
Secretions, Internal: See Endocrine Glands
Sedormid: See Ureides
Senear, F. E.: Lupus erythematosus (extensive), 776
Parkes-Weber syndrome, 602
Psoriasis (pustular) or bacterid? 580
Senear-Usher Syndrome: See Pemphigus
Senility: See Old Age
Septum, Nasal: See under Nose
Serotherapy and Hemotherapy, autohemotherapy for acanthosis rubra lichenoides, 742
autohemotherapy for acanthosis rubra lichenoides; diagnostic comments on article by Antonio Risi, 742
Serum, Convalescent: See Pityriasis rosea
Shaffer, L. W.: Public health problems in control of syphilis, *904
Sharlit, H.: Urinary excretion of indoxyl (as indican) by infants with eczema, *113
Sharp, W. B.: Treatment of ringworm of scalp with gentian violet, *370
Shelmire, B.: Erysipeloid of Rosenbach successfully treated with sulfanilamide, *570
Shelton, J. M.: Progressive pigmentary dermatosis, 952
Shumate, C. A.: Case for diagnosis, 440
Silver, H.: Pemphigus erythematodes (Senear-Usher), 808
Silver, toxicity; argyria, 435, 606, 757
Silvers, S. H.: Dermatitis venenata, case for diagnosis, 177
Simpson, C. A.: Vitamin D in treatment of acne, *835
Skeer, J.: Eruption resembling granuloma annulare, case for diagnosis, 175
Lupus vulgaris, 174
von Recklinghausen's disease, 178
Skeleton: See Bones
Skin, Abnormalities: See also Atrophy; Ectodermal Defect; Pigmentation; etc.
abnormalities; cutis hyperelastica, 1169
abnormalities; Danlos' syndrome, 398
abnormalities; dermatolysis (Ehlers-Danlos syndrome), 1189
abscess; multiple cutaneous abscesses with recurrence in nursling, 388
absorption of roentgen rays; experimental determinations, *87
Atrophy: See Atrophy
bacteriology of normal skin; new quantitative test applied to study of bacterial flora and disinfectant action of mechanical cleansing, 738
Cancer: See Cancer; Epithelioma; Sarcoma; etc.

Skin—Continued
congenital defects of skin, eye and bones, 135
cutaneous absorption; direct technic for demonstrating percutaneous absorption of antigens, *692
cutaneous carbohydrates; normal skin, *193
Diseases: See also Dermatitis; and under names of diseases, as Eczema; Herpes; Mycosis, cutaneous; etc.; and under names of plants and drugs, as Arsphenamines; Rhus; etc.
diseases; hypertherm treatment of cutaneous diseases, *919
diseases; preliminary note on use of "693" (sulfapyridine [2-(paraaminobenzenesulfonamido)-pyridine]) in local applications, 576
diseases; pustular psoriasis or "id"? 186
diseases; role of dietetics in dermatology including discussion of cutaneous manifestations of food allergy, 1112
diseases; role of vitamin C in various cutaneous diseases, *657
diseases; search for filtrable viruses in cutaneous diseases of unknown etiology, *261
diseases; "693" (sulfapyridine [2-(paraaminobenzenesulfonamido)-pyridine]) as local application, 576
excretion of sulfanilamide through skin, *912
Gangrene: See Gangrene
Hemorrhage: See Purpura
hypernephroma with cutaneous metastases, 576
imbibitio lipoidica collageni degenerati cutis, 180, 1193
Inflammation: See Dermatitis
natural color photography of skin; new apparatus for proper illumination, *527
papillomatosis cutis, 935
Pigmentation: See under Pigmentation
rate of ulceration of epitheliomas of skin and lip, *667
senile elastosis and acrodermatitis chronica atrophicans, 168
Syphilis: See Syphilids
temperature measurements in neighborhood of acrocyanosis and lupus vulgaris, 743
Tuberculosis: See Tuberculids; Tuberculosis
Tumors: See under Tumors
Ulcers: See Ulcers
whealing capacity of skin of newborn or young infant; report of experiments, *1029
Slepyan, A. H.: Cheilitis due to deficiency of riboflavin, 1160
Neurodermatitis gigantea, case for diagnosis, 779
Smallpox, eczema vaccinatum, 1137
Smith, E. M., Jr.: Kaposi's sarcoma with lesions of face, 596
Lupus erythematosus, 584
Smith, L. M.: Histologic study of Frei reaction papule, *120
Snails: See Schistosomiasis
Soap, prevention of industrial dermatitis with reference to protective hand creams, soap and harmful role of some cleansing agents, *331
Sobel, N.: Postarsphenamine jaundice, 166
Societies, dermatologic, directory of, 189, 635, 997
examination for certification by American Board of Dermatology and Syphilology, 188, 816, 996
foreign, directory of, 189, 635, 997
international, directory of, 189, 635, 997
local, directory of, 191, 637, 999
national, directory of, 189, 635, 997
sectional, directory of, 190, 636, 998
state, directory of, 190, 636, 998
treatment of psoriasis with concentrated viosterol under auspices of Cincinnati Society of Dermatology and Syphilology, *664
Society Transactions:
American Dermatological Association, Inc., 390
Bronx Dermatological Society, 806, 971
Brooklyn Dermatological Society, 168
Chicago Dermatological Society, 776, 1151
Cleveland Dermatological Society, 139, 604, 798, 1174
Dermatological Conference of Mississippi Valley, 580
Detroit Dermatological Society, 1116
Los Angeles Dermatological Society, 441, 771
Manhattan Dermatologic Society, 132, 610, 938
Minnesota Dermatological Society, 619, 744
New England Dermatological Society, 410, 1125
New York Academy of Medicine, Section of Dermatology and Syphilis, 157, 761, 1180
New York Dermatological Society, 788, 952
Philadelphia Dermatological Society, 179, 444, 625, 980, 1191
Pittsburgh Dermatological Society, 947
San Francisco Dermatological Society, 428, 984, 1141
Sodium Bismuthate: See under Syphilis
dehydrocholate, influence on therapeutic activity of neoarsphenamine; experiments with acute testicular syphilis of rabbits, *838
thiosulfate; action of sulfur-containing compounds in arsenical and mercurial poisoning with review of use of sodium thiosulfate in dermatologic practice and report of experiments on rabbits with sodium thiosulfate, sodium p-sulfhydril phenyl sulfonate and sodium formaldehyde sulfoxylate, *308
Spectacles: See Glasses
Spiegel, L.: Congenital syphilis; juvenile dementia paralytica; interstitial keratitis, 167
Epidermolysis bullosa acquisita, 810
Erb's spastic paraplegia; primary atrophy of optic nerve, 163
Osteoperiostitis of femur, 158
Recent periostitis developing during treatment of late syphilis; iridocyclitis (old) with secondary glaucoma, 157
Spiegler-Fendt Sarcoid: See Sarcoid
Spiller, W. F.: Treatment of ringworm of scalp with gentian violet, *370
Spinal Fluid: See Cerebrospinal Fluid
Spinner, G. J.: Parapsoriasis guttata, 976
Spirochaeta Pallida: See Syphilis
Standish, E. M.: Congenital fragilitas crinium, 1134
Lichen planus, 1128
Lupus erythematosus disseminatus subacutus, 1130
Mycosis fungoides, case for diagnosis, 1126
Pemphigus vulgaris or moniliasis, 1136
Staphylococci, multiple cutaneous abscesses with recurrence in nursling, 388
Stenhouse, E.: Action of sulfur-containing compounds in arsenical and mercurial poisoning with review of use of sodium thiosulfate in dermatologic practice and report of experiments on rabbits with sodium thiosulfate, sodium p-sulfhydril phenyl sulfonate and sodium formaldehyde sulfoxylate, *308
Stern, B.: Generalized dermatophytid after intracutaneous tests with trichophytin in dilutions of 1:30 and 2:0; combination of erysipelas and epidermophytosis, 983
Stewart, C. D.: Cutaneous metastatic carcinoma originating from osteomyelitic cavities, *545
Still's Disease: See Arthritis deformans
Stillians, A. W.: Dermatomyositis, case for diagnosis, 597
Kaposi's sarcoma with lesions of face, 596
Lupus erythematosus, 584
Rosacea-like tuberculosis, 1165

Stokes, J. H.: Case for diagnosis, 181
Stomach, acidity; histamine-like effect of irradiated or inflammatory skin, 741
Stomatitis, dermatostomatitis, 741
erythema exsudativum multiforme (Hebra) with conjunctivitis and stomatitis, *1066
gangrenous; case of noma with recovery following administration of quinine, 579
of vitamin B_2 deficiency treated with nicotinic acid, 575
Stout, O. M.: Bowen's disease of penis, 442
Fixed arsenical pigmentation, 442
Parapsoriasis (en plaque type)? 441
Strauss, A.: Erythema multiforme, pemphigus or dermatitis herpetiformis, 444
Streptococci, spontaneous necrotic dermatitis (hemolytic streptococcus), 414
Sudoriparous Glands: See under Sweat Glands
Sugar, onychia due to handling sugar, 142
Sulfanilamide and Sulfanilamide Derivatives, bullous eruption following sulfanilamide therapy, *61
excretion of sulfanilamide through skin, *912
sensitization reaction to sulfanilamide, 125
Therapy: See Actinomycosis; Chancroid; Dermatitis; Eczema; Erysipelas; Erysipeloid; Lupus erythematosus; Lymphogranuloma; Lymphogranuloma Venereum; Pyoderma gangraenosum; Skin, diseases; etc.
Sulfur, action of sulfur-containing compounds in arsenical and mercurial poisoning with review of use of sodium thiosulfate in dermatologic practice and report of experiments on rabbits with sodium thiosulfate, sodium p-sulfhydril phenyl sulfonate and sodium formaldehyde sulfoxylate, *308
Sulzberger, M. B.: Dermatitis eczematosa (contact type) due to nickel, 815
Whealing capacity of skin of newborn or young infant; report of experiments, *1029
Sunlight: See under Light
Sutherland-Campbell, H.: Superficial noninflammatory lesions of feet; demonstration in sections of fungus of genus Actinomyces, *1023
Sutton's Disease: See Vitiligo
Swartz, J. H.: Scleroderma (progressive type), 419
Sweat and androgenic substance, *654
excretion of sulfanilamide through skin, *912
Sweat Glands, anatomy and pathology of skin appendages; wall of intraepidermal part of sweat duct, 932
chromidrosis; report of case, *379
hereditary ectodermal dysplasia of anhidrotic type associated with syndactylia and lichen nitidus, 946
hidrosadenitis and pyoderma with colitis, 753
pathology of unilateral hyperhidrosis of face of central origin, 742
sudoriparous glands; sweat, *1086
Sweitzer, S. E.: Argyria, 757
Atopic dermatitis, 756
Besnier-Boeck-Schaumann disease, 759
Necrobiosis lipoidica diabeticorum, 757
Sarcoid of Darier and Roussy, 759
Systemic blastomycosis, 758
Sycosis vulgaris, 1116
Syndactylia: See Fingers and Toes, abnormalities
Syphilids, late secondary syphiloderm; syphilitic osteitis? 173
tertiary syphilodermas, 426
Syphilis: See also Chancre; Syphilids; Venereal Diseases; and under names of organs and regions, as Cardiovascular System; etc.
acute syphilitic transverse myelitis; clinical study and report of case, *1078
ainhum or periarteritis, 443
caution in arsphenamine therapy (report of case of purpura haemorrhagica following treatment with neoarsphenamine), 1111
clavicle sign of late congenital syphilis; review of literature and report of 6 cases, *1060
Syphilis—Continued
combination courses of bismuth administration, 125
complications due to arsenical therapy in syphilitic pregnant women; report of 7 maternal deaths, 128
congenital, 148
congenital; juvenile dementia paralytica; interstitial keratitis, 167
death following mapharsen therapy, 384
diagnosis from single drop of blood by Chediak-Dahr method, 741
drug eruption, lichen planus or pityriasis lichenoides et varioliformis acuta, 765
effect of adding chlorobutanol to bismuth subsalicylate in oil injected intramuscularly, *380
effect of bismuth therapy on latent plumbism; preliminary report, *38
elephantiasis of lips and of male genitalia with reference to syphilis and lymphogranuloma venereum as etiologic factors, *11
erythema of ninth day following bismuth therapy for, *1076
erythroderma in course of bismuth therapy, 933
experimental research into curative effect of physically induced hyperthermy, 742
facial paralysis due to early syphilis of central nervous system; granuloma inguinale, 154
Fuchs reaction in, 936
glossitis rhombica mediana; interstitial atrophic syphilitic glossitis, 979
herpes zoster occurring during treatment for, *929
influence of sodium dehydrocholate on therapeutic activity of neoarsphenamine; experiments with acute testicular syphilis of rabbits, *838
late secondary syphilis with pyogenic infection and syphilitic dactylitis, 449
late secondary syphiloderm; syphilitic osteitis? 173
latent, serologic verification test in diagnosis of, *817
laws requiring premarital and pregnancy tests for syphilis; reasons for and against, 1112
livedo racemosa in syphilitic patient, 152
meningomyelitis syphilitica with muscular atrophy, 158
mucocutaneous relapse or reinfection, 1177
occipital puncture in hands of dermatologist, 577
or carcinoma of tongue, 979
or lupus vulgaris? 426
overtreatment in, 1112
perforating gumma of soft palate and nasal septum; asymptomatic neurosyphilis, 751
production of positive serologic reactions in rabbits and subsequent reactions of serologically-altered animals to inoculation with Spirocheta pallida, 387
prognosis of, 126
protective antibodies in serum of syphilitic rabbits, 386
public health problems in control of, *904
pustular rupial syphilis (?), 980
recent periostitis developing during treatment of late syphilis; iridocyclitis (old) with secondary glaucoma, 157
simplified complement fixation technic for diagnosis and treatment; its sensitivity and specificity, *32
simplified technic for administering old arsphenamine, 384
spontaneous fever therapy in neurosyphilis, 127
superinfection in; report of possible case, *1108
syphilitic atrophy of optic nerve, 971
syphilitic leukoderma following secondary papular eruption, 814

Syphilis—Continued
syphilitic macular atrophy, 151
tertiary, 392
tertiary, and syphilitic macular atrophy, 811
tertiary; aneurysm of ascending portion of aorta, 161
tertiary; atrophic arthritis with subcutaneous nodules and tertiary syphilis, 143
tertiary; gumma of larynx, 979
tertiary; multiple tuberculous gummas in course of primary infection, 1115
tertiary, or moniliasis, 767
tertiary; transverse myelitis following administration of bismuth compound, 160
treated; lymphogranuloma venereum? 621
with negative serologic changes; exfoliative dermatitis, 391
with reference to incidence in relationship to age groups and status of therapy at date of infection, 129
Syphiloderm: See Syphilids
Syringobulbia: See Medulla Oblongata
Syringomyelia, lumbosacral type, 433

Tabes Dorsalis, juvenile, 154
juvenile; primary atrophy of optic nerve, 447
tabes (Charcot knee; atrophy of optic nerve; chorioretinitis), 171
tryparsamide dermatitis, 428
Talbott, J. H.: Pemphigus; experimental studies on 34 patients, *359
Role of vitamin C in various cutaneous diseases, *657
Tattooing, tattoo marks; result of attempts to remove them, 619
Tauber, E. B.: Hypertherm treatment of cutaneous diseases, *919
Taussig, L. R.: Necrobiosis lipoidica diabeticorum, case for diagnosis, 1143
Urticaria pigmentosa, 992
Teeth, fistulas from dead teeth simulating dermal epithelioma, *378
Telangiectasis: See also Angioma; Purpura annularis telangiectodes
hereditary familial; case for diagnosis, 1189
hereditary hemorrhagic telangiectasia, 986
macularis eruptiva perstans? 994
nevus or hereditary familial telangiectasia, 938
Templeton, H. J.: Atrophic process of glans penis with epithelioma, 432
Epidermolysis bullosa congenita, 434
Epidermolysis bullosa neonatorum, 993
Hydroa aestivale, 432
Laurence R. Taussig, 930
Oriental sore, 970
Purpura annularis telangiectodes, 437
Telangiectasia macularis eruptiva perstans, case for diagnosis, 994
Testicles, influence of sodium dehydrocholate on therapeutic activity of neoarsphenamine; experiments with acute testicular syphilis of rabbits, *838
Testosterone Propionate: See Androgens
Thiamin Chloride: See Vitamins, B
Thiocyanate dermatitis, 573
Thomas, C. C.: Virus infection resembling herpes simplex, 179
Thrombocytopenia: See Purpura haemorrhagica
Thyroid: See Myxedema
Thyrotoxicosis: See Goiter, exophthalmic
Ticks, wood tick simulating pedunculated tumor, *887
Tilden, I. L.: Relapsing febrile nonsuppurative panniculitis; report of 2 cases, *681
Tinea: See also Ringworm
barbae, dermatophytosis and tinea corporis due to Trichophyton purpureum, 938
capitis; kerion celsi, 748
capitis; kerion celsi; trichophytid, 748
corporis, dermatophytosis and tinea barbae due to Trichophyton purpureum, 938
cruris and corporis, probably due to Trichophyton purpureum, 1146
Tinea—Continued
Decalvans: See Alopecia areata
Imbricata: See Ringworm
versicolor; Malassezia furfur, cause of tinea versicolor; cultivation of organism and experimental production of disease, *253
Tobin, W. W.: Acarophobia and tumors due to camphor in oil, 601
Arsenical pigmentation and keratoses first manifested in patient at age of 6 years, 778
Lichen planus, 787
Lupus erythematosus with acrosclerosis, 1162
Recklinghausen's disease, 593
Toe-Nails: See Nails
Toes: See Fingers and Toes
Tolmach, J. A.: Arsenical dermatitis with pigmentation and alopecia, 761
Generalized Trichophyton purpureum infection simulating dermatitis herpetiformis; report of case, *732
Tolman, M. M.: Discoid lupus erythematosus with dissemination, 419
Lupus erythematosus? 425
Lupus erythematosus or lichen planus? 411
Tongue, atrophic glossitis, case for diagnosis, 769
cancer, or syphilis, 979
extragenital lymphogranuloma venereum on tongue and cervical nodes, 576
glossitis rhombica mediana; interstitial atrophic syphilitic glossitis, 979
lingua nigra, 390
lupus vulgaris; ulcers of tongue (unknown cause); osteomyelitis of right humerus, 624
report on case of ulcer of, 798
ulcerated lichen planus of, 933
Tooth paste, cheilitis and dermatitis from, *892
Torrey, F.: Atopic dermatitis, 1149
Tuberculous granuloma of nose, case for diagnosis, 435
Toxicodermas: See Dermatitis, venenata; Drugs, eruptions
Traub, E. F.: Blood iodine of patients with acne vulgaris, *506
Bourneville-Pringle syndrome, 791
Case for diagnosis and suggestions for therapy, 963
"Common mole"; its clinicopathologic relations and question of malignant degeneration, *214
Congenital fragilitas crinium, 791
Granuloma annulare (left leg), 792
Lichen planus following gold therapy, 763
Lichen planus with annular lesion on scalp, 796
Lymphatic leukemia with leukemids, 964
Necrobiosis lipoidica diabeticorum, 959
Pemphigus erythematodes, case for diagnosis, 791
Pemphigus or lupus erythematosus bullosus, 797
Trichomycosis: See Hair, diseases
Trichophytid: See Trichophytosis
Trichophytin: See Dermatitis herpetiformis; Trichophytosis
Trichophyton purpureum (Bang), cultural variations with discussion of recognizable features, *895
purpureum, dermatophytosis, tinea barbae and tinea corporis due to, 938
purpureum; generalized Trichophyton purpureum infection simulating dermatitis herpetiformis; report of case, *732
purpureum, tinea cruris and corporis probably due to, 1146
Trichophytosis: See also Mycosis
delayed reaction to trichophytin, 945
tinea capitis; kerion celsi; trichophytid, 748
Trichophyton purpureum infection, 989
Trichorrhexis Nodosa: See Hair, diseases
Triethanolamine: See Ethanolamine
Trophedema: See Edema
Tryparsamide dermatitis, 428
nitritoid reaction to; report of case, *690

Tubercle Bacilli: See under Tuberculosis
Tuberculids: See also under Tuberculosis
papulonecrotic, case for diagnosis, 441
papulonecrotic, or granuloma annulare, 961
rosacea-like, of Lewandowsky, 744, 1175
rosacea-like, of Lewandowsky; pulmonary tuberculosis, 608
Tuberculin: See under Tuberculosis
Tuberculoderm: See Tuberculids
Tuberculoma of palate and right angle of mouth; miliary pulmonary tuberculosis, 1179
Tuberculosis: See also under Bones; Mouth; Tongue; etc.
and acne, 936
atrophic arthritis with subcutaneous nodules and pulmonary tuberculosis, 144
distinctive type in Negro associated with tuberculous keratouveitis, 445
gold therapy; pathogenesis of hyperkeratoses in gold dermatitis, 578
granuloma annulare; advanced bilateral cavitating pulmonary tuberculosis, 604
lupus erythematosus with tuberculin reaction, 942
lupus vulgaris of face, tuberculosis of buccal mucous membranes, pharynx and larynx; pulmonary tuberculosis, 1179
micronodular tuberculids, 1115
miliary cutaneous tuberculosis associated with miliary pulmonary tuberculosis of chronic evolution, 575
multiple tuberculous gummas in course of primary infection, 1115
of skin; case for diagnosis, 977
pulmonary; rosacea-like tuberculid of Lewandowsky, 608
pulmonary; scrofuloderma, 624
rosacea-like, 1165
tuberculoma of palate and right angle of mouth; miliary pulmonary tuberculosis, 1179
tuberculous granuloma of nose; case for diagnosis, 435
verrucosa cutis, 779
Tuberculum Dolorosum: See Tumors
Tumors: See also Angioma; Arrhenoblastoma; Cancer; Epithelioma; Hemangioma; Histiocytoma; Lymphangioma; Lymphoblastoma; Melanoma; Papilloma; etc.; and under names of organs and regions, as Brain; Face; Ovary; Palate; etc.
acarophobia and tumors due to camphor in oil, 601
Danlos' syndrome, 398
dermatolysis (Ehlers-Danlos syndrome), 1189
metastases of scalp simulating turban tumors, *639
oil tumors; case for diagnosis, 1153
painful subcutaneous tubercle (tuberculum dolorosum), 1110
unusual location of glomus tumor; report of 2 cases, 130
wood tick simulating pedunculated tumor, *887
Turell, R.: Treatment of pruritus ani by tattooing with mercury sulfide, *521
Twining, H. E.: Lichen planus or dermatitis medicamentosa from bismuth, 627
Universal depigmentation of skin, with areas of hyperpigmentation, 444
Tyroglyphus: See under Mites

Ulcers, chronic, of back; case for diagnosis, 952
chronic, of leg associated with congenital hemolytic jaundice, 128
hemolytic jaundice with ulceration of skin, 408
lupus vulgaris; ulcers of tongue (unknown cause); osteomyelitis of right humerus, 624
multiple epitheliomas; ulceration following use of "cancer paste," 1174
rate of ulceration of epitheliomas of skin and lip, *667
trophic, of face and neck due to syringobulbia, 153
Varicose: See Varicose Veins
Ultraviolet Rays: See also Light
actinic atrophy; case for diagnosis, 791
influence of ultraviolet irradiation on excretion of sex hormones in male, 386
roentgen dosage in dermatology expressed in international roentgens, *1
Urbach, E.: Erythema annulare rheumaticum (Lehndorff-Leiner); report of case, *515
Generalized dermatophytid after intracutaneous tests with trichophytin in dilutions of 1:30 and 2:0; combination of erysipelas and epidermophytosis, 983
Imbibitio lipoidica collageni degenerati cutis, 180, 1193
Lymphogranulomatosis (Hodgkin's disease); treatment with sulfanilamide, 181
Parapsoriasis lichenoides chronica, 1197
Urea: See under Verruca
Ureides, purpura haemorrhagica following use of sedormid; 2 attacks in 1 person, 129
Urethra, inguinal adenopathy of probable gonococcic origin (urethritis), 932
melanoma of, 1110
Urine: See also Urination
excretion of indoxyl (as indican) by infants with eczema, *113
influence of ultraviolet irradiation on excretion of sex hormones in male, 386
urinary excretion of estrogenic substances after administration of testosterone propionate, 386
Urticaria due to heat, 949, 1191
from cold, 439
pigmentosa, 146, 610, 798, 992
relief of arthritic symptoms following urticaria, 1113
vitamin C in, *660
whealing capacity of skin of newborn or young infant; report of experiments, *1029
Usher, B.: Danlos' syndrome, 398
Kraurosis vulvae with leukoplakia, 395
Leukoplakia vulvae, 396
Mycosis fungoides, 403
Neurodermatitis, 395
Pemphigus (Senear-Usher syndrome), 405
Prurigo (Hebra), 406
Usher-Senear Syndrome: See Pemphigus
Uvea, distinctive type of tuberculosis in Negro associated with tuberculous keratouveitis, 445

Vaccinia: See Smallpox
Vagina, granuloma inguinale of vaginal wall and rectum with profuse hemorrhage, 801
Varicose Veins, effective treatment of varicose ulcers of leg, *530
Parkes-Weber syndrome, 602
Variola: See Smallpox
Varix: See Varicose Veins
Veins, Intermittent Compression: See Extremities, blood supply
Varicose: See Varicose Veins
Venereal Diseases: See also Chancre; Chancroid; Syphilis
laws requiring premarital and pregnancy tests for syphilis; reasons for and against, 1112
syphilis and law with discussion of false positive blood serologic test, 127
Verruca, Tuberculous: See under Tuberculosis vulgaris; use of urea (carbamide) in treatment of warts, *736
Vesication, recurrent vesicopustular eruption on extremities, 394
Vieira, J. P.: Pemphigus foliaceus (fogo salvagem); endemic disease of state of São Paulo (Brazil), *858
Viosterol: See Psoriasis
Viruses: See also Herpes; etc.
filtrable, search in cutaneous diseases of unknown etiology, *261
inquiry into cause of pemphigus; is it virus disease? *78
virus infection resembling herpes simplex, 179

Viscera, Boeck's sarcoid, 573
herpes zoster and its visceral manifestations, 383
Vitamins: See also Pellagra; etc.
B; cheilitis due to deficiency of riboflavin, 1160
B; herpes zoster and vitamin B_1, 1113
B; stomatitis of vitamin B_2 deficiency treated with nicotinic acid, 575
C, role in various cutaneous diseases, *657
D in treatment of acne, *835
D; pemphigus of long duration in mouth with acute exacerbation on skin; improvement following large doses of vitamin D, 630
generalized lupus erythematosus, vitamin deficiency (pseudopellagra), dermatomyositis or Addison's diseases, 185
in cosmetics, *74
vitamin therapy in dermatology with reference to vitamin D in treatment of acne and of diseases due to altered usage of calcium, *842
Vitiligo, depigmentation at site of chronic contact eczema, 621
leukoderma, 1132
leukoderma acquisatum centrifigum (Sutton), 771, 1190
leukoderma following dress shield dermatitis, 1141
leukoderma (occupational), 587
syphilitic leukoderma following secondary papular eruption, 814
with superimposed pellagra, 625
Vörner's Disease: See Heloderma
Vogel, H. R.: Onychomycosis due to Monilia albicans, 951
Vulva, Diphtheria: See under Diphtheria
Esthiomene: See Lymphogranuloma Venereum
kraurosis vulvae with leukoplakia, 395
leukoplakia vulvae, 396
lymphangiectasia of vulva with edema of legs, 146

Waisman, M.: Dermatomyositis; study of 40 cases, *1001
Walzer, A.: Cutaneous absorption; direct technic for demonstrating percutaneous absorption of antigens, *692
Dermatolysis (Ehlers-Danlos syndrome), 1189
Senile elastosis and acrodermatitis chronica atrophicans, 168
Tabes (Charcot knee; atrophy of optic nerve; chorioretinitis), 171
Warren, L. H.: Leukoderma (occupational), 587
Warts: See Verruca
Wassermann Reaction: See also Syphilis
temporarily positive Kahn and Wassermann reactions in infectious mononucleosis; report of case, 129
Way, S. C.: Sudoriparous glands; sweat, *1086
Wayson, J. T.: Acute arthritis associated with myocarditis simulating acute lupus erythematosus; report of case, *1053
Weber-Rendu-Osler's Disease: See Telangiectasis
Weber-Christian's Disease: See Panniculitis
Webster, J. R.: Arsenical pigmentation and keratoses first manifested in patient at age of 6 years, 778
Mycosis fungoides, 581, 582
Weiner, A. L.: Disseminated lupus erythematosus treated by sulfanilamide; report of 4 cases, *534
Pyoderma gangraenosum treated with sulfanilamide; report of case, *711
Wheal: See Urticaria
White, C. J.: Psoriasis pustulosa, case for diagnosis, 1157
White Spot Disease: See Scleroderma
Wien, M. S.: Cheilitis due to deficiency of riboflavin, 1160
Neurodermatitis gigantea, case for diagnosis, 779
Wilson, W. D.: Rate of ulceration of epitheliomas of skin and lip, *667
Wise, F.: Acanthosis nigricans, 797
Lepra with juxta-articular nodules, 789
Parapsoriasis, 610
Senear-Usher pemphigus or dermatitis medicamentosa, 795
Urticaria pigmentosa, 610
Wolcott, G. L.: Dangers in use of cosmetics, *64
Wolff, M. J.: Dermatitis herpetiformis perstans, 773
Multiple benign epitheliomatosis, 774
Woolhandler, H.: Cutaneous metastatic carcinoma originating from osteomyelitic cavities, *545
Workmen's Compensation and Insurance: See Industrial Diseases
Wright, C. S.: Hypertrophic nevus of scalp, face and oral mucosa, 1191

Xanthoma, 622
diabeticorum chiefly limited to palms, 139
juvenile, 586
juvenile disseminated xanthomatosis, 1134
juvenile xanthomatosis (possible Hand-Schüller-Christian disease), 585
mollusciforme et generalisatum in infant, 743
tuberosum multiplex, 617
Xanthomatosis: See Schüller-Christian Syndrome; Xanthoma

Yang, K. L.: Clavicle sign of late congenital syphilis; review of literature and report of 6 cases, *1060

Zeisler, E. P.: Dermatitis with depigmentation (pellagra?), 1152
Juvenile xanthomatosis (possible Hand-Schüller-Christian disease), 585
Subacute disseminated lupus erythematosus, 1161
Zoster: See Herpes zoster
Zugerman, I.: Lupus erythematosus, 980
Pustular rupial syphilis (?), 980

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